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# PNEUMOCEPHALUS (INTRACRANIAL PNEUMATOCELE OR AEROCELE) *

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For at least a century and a half it has been known that under certain pathologic conditions—infection and trauma—air can pass through a break in the outer wall of a cranial air sinus into the scalp and, gradually traveling in the loosely attached subaponeurotic layer, form air tumors which at times may be of tremendous size (fig. 1). The two sources of these extracranial aerogenous tumors (recorded as pneumatoceles or aeroceles) have been (1) the mastoid cells and (2) the frontal sinus, the former being much more frequent.[1] It has also long been known that steady pressure on such gaseous tumors caused them to disappear, the air being forced through the eustachian tube or frontal sinus. Swallowing or coughing would again cause the occipital, and coughing the sincipital, variety quickly to reappear. A number of cases of the occipital type have been cured by treating the opening which leads from the sinus into the scalp with iodine or other stimulant, while others have healed spontaneously as granulation tissue gradually choked the fistulous tract.

It is safe to presume that infections and fractures must also have opened the inner wall of the air cells, and at times the dura, and under proper conditions must have passed through these defects not only between the bone and the dura but at times into the cranial chamber. Intracranial air tumors, however, giving no such simple signs of their presence as those of the extracranial variety, have existed without clinical recognition until recent years. The roentgenogram has now made the detection of intracranial air almost as easy and at least as certain as the direct observation of an extracranial pneumatocele. Apparently only once has the presence of intracranial air that has developed during life been satisfactorily demonstrated without the use of the roentgen ray. This remarkable exception was observed by Chiari [2]

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1. Wernher: Pneumatocele Cranii, Deutsche Ztschr. f. Chir. **3**:381, 1873.

2. Chiari, H.: Ueber einen Fall von Luftansammlung in den Ventrikeln des menschlichen Gehirns, Prag. Vierteljahrsschr. f. Heilk. **5**:383, 1884.

in 1884. At necropsy he not only demonstrated that air filled a large cavity in the frontal lobe and the ventricular system as well, but he located a tiny opening leading from an ethmoid cell to the frontal cavity and explained the ingress of air by the explosive force of sneezing (fig. 2). Chiari was confident that no postmortem factors could explain the presence of air and concluded it had surely been present during life. Chiari's brilliant report antedated by many years the advent of roentgenography and by nearly thirty years Luckett's [3] case (1913), this being the first instance in which air was detected by the use of the roentgen ray.

The following year, 1914, Wolff [4] independently discovered air in a German case. Twenty-five cases have been compiled from the literature to date, eleven from the United States, eleven from Germany, two from France and one from Italy. To these we add three more, all of which have been seen in a period of eight months.

The mere annexation of these additional cases is of little concern but the need for better recognition of this condition—symptoms, etiology, pathology and treatment—is best attested by the fact that in only a single instance [5] has intracranial air been suspected before its disclosure at necropsy, operation or by roentgenography. The occurrence of our two cases within a period of six months also indicates that this condition merits consideration in the differential diagnosis of cerebral lesions. Treatment moreover is dependent on an early and accurate diagnosis.[6]

There are two sources of intracranial air: (1) a break in the wall of the skull, through which air is forced from the exterior, and (2) the product of gas producing organisms after their entrance into the brain.

After the initial cranial defect, air can be forced into the brain only through an opening in one of the paranasal sinuses or the mastoid cells, for here alone can the air be concentrated by swallowing, coughing or

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3. Luckett, W. H.: Air in the Ventricles of the Brain Following a Fracture of the Skull: Report of a Case, Surg., Gynec. & Obst. **17**:237, 1913. Stewart, W. H.: Fracture of the Skull with Air in the Ventricles, Am. J. Roentgenol. **2**:83-97, 1913-1914.

4. Wolff: Luftansammlung im Rechten Seitenventrikel des Gehrins (Pneumocephalus), München. med. Wchnschr. **16**:899, 1914.

5. Wodarcz, A.: Zur Kosnistik der intracranieller Pneumatozele, München. med. Wchnschr. **62**:968, 1915.

6. All of the German cases have been reported under the title intracranial "pneumatocele," many of the American and one of the French as "aerocele." Though equally correct, the former is to be preferred because it maintains an association with the long established "extracranial" pneumatocele. However, as the intracranial air is at times not confined to a "tumor" but may be definitely distributed throughout the ventricular system or subarachnoid space or multiple isolated pockets may exist within the cerebral substance, it seems preferable to forego both of these appellations and substitute "pneumocephalus," which connotes only the presence of intracranial air.

sneezing under a pressure greater than that within the cranial chamber. The bony wall of a sinus can be opened by a fracture, an operation, an infection or by the persistent pressure of a tumor or of the dilated third ventricle of hydrocephalus. In one of the cases here reported, the air entered through a fracture of the frontal bone; in a second, through a fracture through the temporal bone, and in the third, it probably followed an operative defect of the mastoid cells, though the possibility of a bacterial gas product cannot be absolutely eliminated.

## REPORT OF CASES

CASE 1.—A man, aged 70, referred by Dr. H. I. Thomson of St. Petersburg, Fla., was brought to the Johns Hopkins Hospital, Feb. 20, 1925, in a semi-comatose condition. With strong stimulation by supra-orbital pressure it was possible to get a few disconnected and irrelevant sentences, which indicated complete disorientation as to time, place and persons.

The following story was obtained from relatives: January 6, (forty-five days before admission) he was struck by an automobile and thrown to the pavement, striking his right forehead; he was unconscious for only a few minutes. During a stay of several days in the hospital he was irrational much of the time, but in the second week his condition improved so much that he was discharged from the hospital. Much of the third week was spent at home in bed, but the following week he was up and about most of the time and transacted some business. His wife, however, noticed that he was careless about financial details, about his clothes and other matters, in all of which he had formerly been particular; but throughout the fourth week there seemed a steady improvement. One afternoon, about a month after the accident, the patient was found holding his head in his hands; he said that he would go crazy if he could not get some relief from a terrific headache. During the next few days he was dazed most of the time and again he was taken to the hospital by Dr. Thomson (February 5). He was then in a stupor and, when with difficulty he could be aroused, he was always irrational. Three days later (February 8) he seemed much better, though his condition varied greatly from time to time during the day; on the whole he was much more conscious and often was quite rational. A week before his admission to the hospital (February 11) he had a severe spell of trembling over the whole body which was said to have been more pronounced in the arms and head; a roentgenogram taken February 11 showed a large amount of air in both lateral ventricles (fig. 4). Again he became irrational and raved on various subjects, jumping from one to another without reason. During much of the time he had to be restrained. His pulse was slow, from 50 to 70; the temperature never rose above 99 F.

He was in this condition on arrival in Baltimore except that restraint was no longer necessary and he was decidedly difficult to arouse, strong supra-orbital pressure being necessary to bring any response. His general physical condition was good; he was stronger and younger than his years would indicate. To the right of the midline the supra-orbital ridge was definitely depressed, though the skin was not broken, nor was this region noticeably tender. There were no extra-ocular palsies nor signs of unilateral motor or sensory disturbance. The eye grounds showed neither engorgement of the veins nor swelling of the optic disks. Examinations of the remaining cranial nerves and regions of the brain were negative. Apparently there had been no disturbance

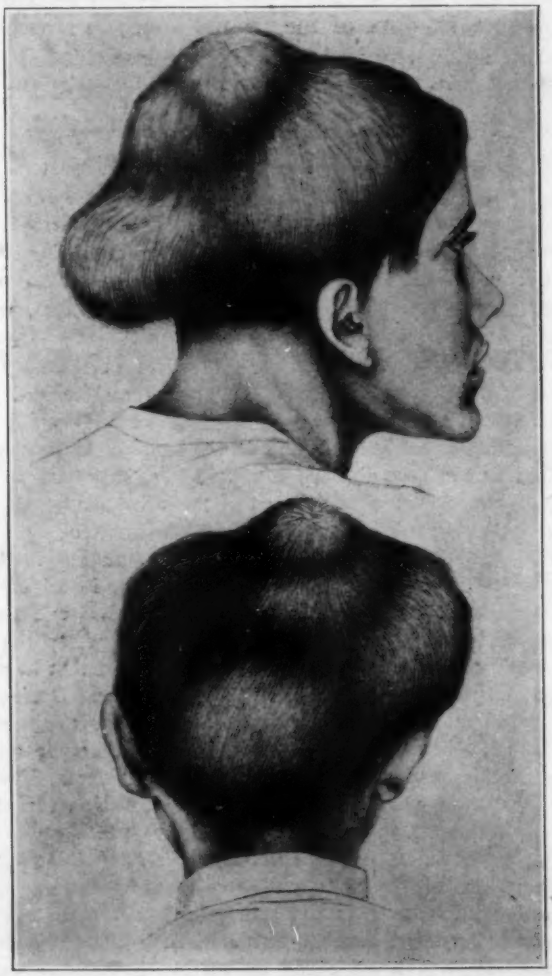

Fig. 1.—Occipital extracranial pneumatocele; this air could be evacuated into the pharynx by pressure and again returned with swallowing (from Wernher: Deutsche Ztschr. f. Chir. 3:381, 1873). The only difference between an extracranial and an intracranial pneumatocele is the properly situated opening in the dura.

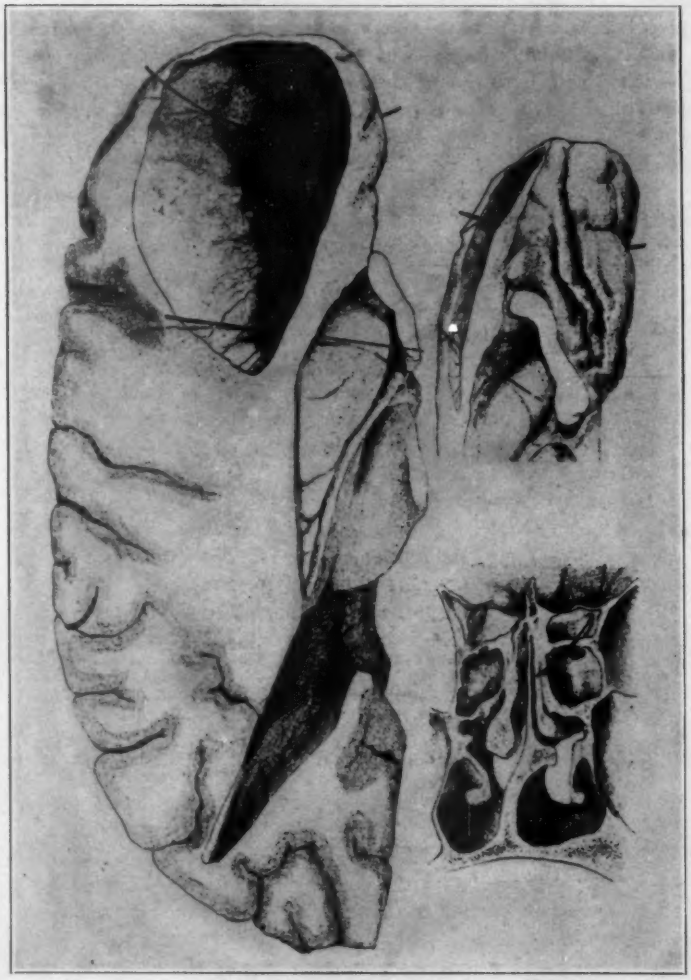

Fig. 2.—Intracranial pneumocephalus (from Chiari: Ztschr. f. Heilk. **5**:391, 1884); this remarkable case, antedating by many years the advent of roentgenography, seems to be the only case of pneumocephalus disclosed without the use of the roentgen ray; the arrows indicate the opening between the ethmoid sinus and the frontal defect, and between the frontal defect and the lateral ventricle; except for the sinus involved the pathologic findings in this case are practically identical with those of our own (case 1).

of the gait, and he had not complained of diplopia or cervical rigidity. The reflexes were equal and normally active on the two sides. Plantar stimulation gave plantar flexion. The pulse was 50; temperature, 100 F. The leukocytes totaled 8,000. The systolic blood pressure was 110, diastolic 70.

It was our impression that this lesion was probably a chronic subdural hemorrhage. Doubtless this diagnosis was entertained the more because of another patient who had just been operated on and who had a strikingly similar story. It was quite clear that there was greatly increased intracranial pressure because of his stupor and bradycardia (with normal urinary findings), the negative optic disk notwithstanding. An abscess seemed improbable because fever had been absent throughout his illness; there had never been cervical rigidity and the leukocyte count was now normal. The possibility of a pneumatocele was not considered. In the absence of any localizing signs or symptoms, the

Fig. 3 (case 2).—Lateral view; the upper group of arrows point to the air in the anterior horn of the lateral ventricle; the air reached the ventricle through a perforation in an air containing abscess of the temporal lobe; the lower arrows indicate two separate pockets of air in the abscess itself. While we believed that the air was forced through the operative mastoid defect into the temporal lobe abscess, the possibility that the air is of bacterial origin cannot be excluded.

depressed fracture was looked on as probably indicating the side of the presumed subdural hemorrhage. Solely on this finding exploration of the right hemisphere was determined.

Being so nearly in coma he was at once prepared for operation. Fortunately, though merely as a matter of routine, stereoscopic roentgenograms were taken en route to the operating room. As nothing significant was expected from the roentgen-ray examination, development of the plates was not awaited. The first intimation of the character of the intracranial lesion was therefore at the operation.

A frontoparietal bone flap of usual size and form was turned down under local anesthesia. The dura was extremely tense and when incised the brain herniated to the maximum degree (fig. 7). There was no subdural hemorrhage but the bulging brain at once indicated a subcortical lesion in the frontal lobe which, being very soft, protruded like a ball from the level of the firmer parietal and occipital lobes. The frontal convolutions were extremely wide and pale (fig. 7). The great pressure from within the brain had squeezed

Fig. 4 (case 1).—Anteroposterior view, showing air in anterior horn of both lateral ventricles following fracture of frontal bone, three weeks after accident.

nearly all of the blood supply from the thinned cortex. The sulci were obliterated and entirely free of fluid. Even the larger vessels of the frontal lobe were scarcely visible. We fully expected a big subcortical hemorrhage but when the greatly thinned frontal lobe was incised a strong puff of air followed. The protruding frontal lobe at once collapsed and instead of the tremendous cerebral hernia which had been present only a moment before, the

brain was now sunken far below the dural margins (fig. 8). The frontal lobe was now a great cavity easily as large as a goose egg; the cortex was a mere shell varying from 0.5 to 1 cm. in thickness. Roentgenograms were immediately sent for; these verified the operative findings of intracranial air (figs. 5 and 6).

Inspection of the great cavity showed the lining to be everywhere smooth, glistening and avascular. There was no fluid within the cavity. At first

Fig. 5 (case 1).—Anteroposterior view at time of operation and eleven days after figure 4; the amount of air has increased and the size of the ventricles is therefore much greater.

nothing unusual was seen, but on the inner (mesial) wall beneath a fold that was readily pushed aside with the spatula, a small oval opening (about 0.5 cm. long) was brought into view. This foramen led directly into the anterior horn of the right lateral ventricle (the upper outer wall) and explained the entrance of air into the ventricles (fig. 8). There was no visible fluid in this part of the

ventricle. The foramen of Monro and the choroid plexus were easily seen through the opening. In the anterior tip of the big frontal defect a second opening of smaller size was superimposed on a dural defect. A probe was passed through this opening but it could not find a corresponding defect in the skull, i. e., the probe did not pass into the frontal sinus. Around this dural defect the cortex was rather firmly adherent over an area probably 4 by 4 cm., the adhesions being most dense surrounding the fistula (fig. 8).

The frontal lobe was then dissected free of the adherent dura; the denuded dural opening was again evident but again there was no direct tract into the frontal sinus. There could be no doubt that despite the failure of the probe to enter the frontal sinus, the air must have entered the cerebral defect from that source—an explanation to appear later. Our object in freeing the frontal lobe from the dura was to close the opening from the inside but the great depth

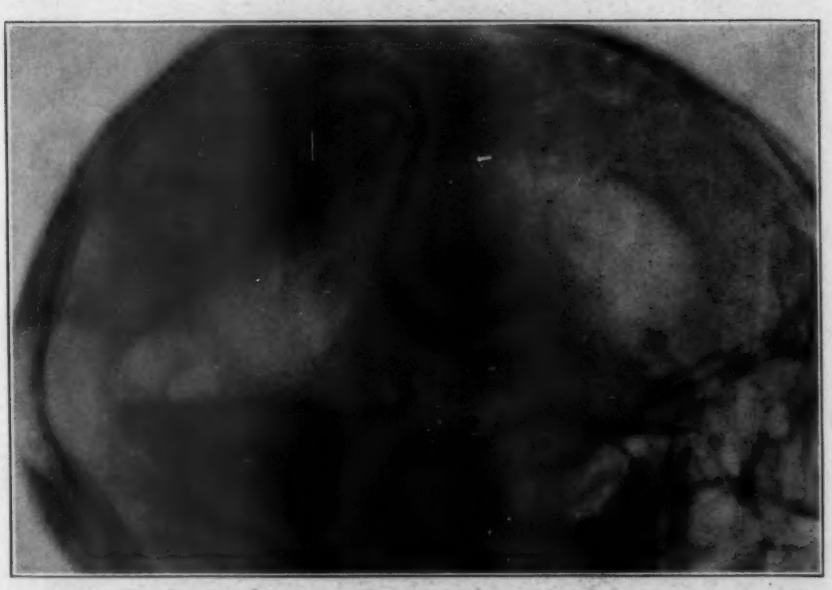

Fig. 6 (case 1).—Lateral view of head showing air in left lateral ventricle; had the head been taken with the left side on the roentgen-ray plate, the right frontal defect, which is absent in this plate, would have been filled; the ventricle is tremendously dilated.

of the wound and the close contact between dura and bone made this impractical. The frontal defect was then filled with Ringer's solution, the dura carefully sutured, the bone flap replaced and the wound closed.

A second incision was then made through the shaved eyebrow in order to expose the depressed fracture in the right supra-orbital region. The fragment of bone measuring about 2 by 2 cm. was depressed to a depth varying from scarcely a millimeter on the outer side to probably 5 mm. mesially (fig. 9). The entire fragment was pried from its bed by a periosteal elevator. The line of fracture had passed through the frontal sinus on the mesial side only.

A semilunar tear in the dura (about 3 or 4 mm. in length) was exposed when the bone had been removed. It was situated not directly beneath the

line of fracture but at a point corresponding with the center of the depressed piece of bone (fig. 10). As shown in figures 12 and 13, it was necessary for the air from the frontal sinus to be deflected around the posterior surface of the bone before entering the dural defect. This devious passage was in effect a potential valve. The pressure from sneezing or coughing would easily push the dura backward and force the air directly through the dural fistula into the frontal cavity but the increased intracranial pressure at once sealed the dura against the frontal bone and thus precluded the exit of air.

A small piece of fascia lata was placed over the dural defect and sutured to the dura by a row of silk sutures; the opening was covered with a good margin to spare (fig. 11). This effectually prevented the air from again entering the cranial chamber.

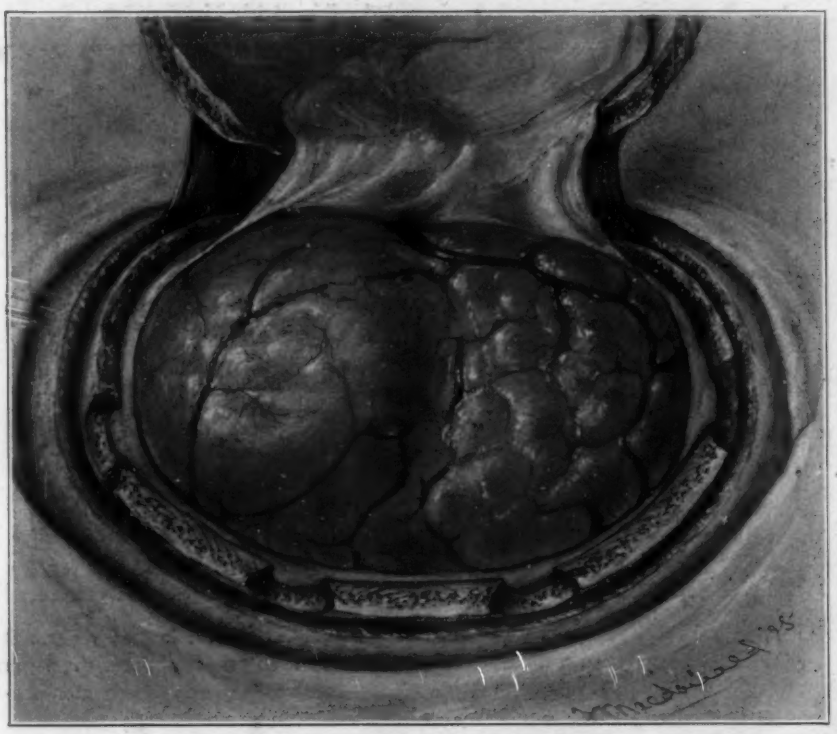

Fig. 7 (case 1).—Brain as exposed at operation; the frontal lobe was a bulging sac of air; the convolutions and the cortical vessels were nearly obliterated.

The posterior wall of the frontal sinus was removed in part in order to preclude the accumulation of air extradurally—a seeming possibility—with an inadequate opening for the escape of the air. Healing was by first intention.

The postoperative recovery was rapid and uneventful. When a roentgenogram was taken on the patient's discharge from the hospital, March 3, 1925, thirteen days after the operation, the air had completely disappeared. The frontal defect was filled with Ringer's solution; no other effort was made to replace the air that still remained within the cranial chamber. Experiences with artificial

cerebral pneumography[7] have shown that air filling the ventricular system will be completely absorbed in less than twenty-four hours, if the ventricular channels are not blocked. As the air is absorbed the large cerebral defect is automatically filled with cerebrospinal fluid of ventricular origin and at once assumes the aspect of a healed cavity.

In retrospect it is evident that an accurate diagnosis would have made the problem of cure much easier and safer for the patient, i. e., merely closure of the dural opening and possibly enlargement of the posterior

Fig. 8 (case 1).—Incision of the cortex released the air and caused the frontal lobe to collapse; the opening in the dura is shown by the point of the probe; immediately superimposed (arrow) is the opening into the frontal pneumatocele.

wall of the frontal sinus. Had we been even less positive that the lesion was on the side of the fracture—and our reasons were not too free from the possibility of error—a ventricular estimation test or an air injection would have been in order and the diagnosis might have resulted from

7. Dandy, W. E.: Ventriculography Following the Injection of Air Into the Cerebral Ventricles, Ann. Surg. **68**:5 (July) 1918; Roentgenography of the Brain After the Injection of Air Into the Spinal Canal, Ann. Surg. **70**:397 (Oct.) 1919.

the air escaping from the uppermost ventricle, but there is a fair chance that the escape of air might not have been detected. The mistaken diagnosis fortunately permitted an inspection of the anatomic changes of this rare condition and afforded an opportunity to interpret the sequence of events.

CASE 2.—A man, aged 29, entered the medical service of the Johns Hopkins Hospital in a state of semicoma, with a temperature of 107.2, pulse 110. One week before admission he came home from work complaining of severe headache and pain in the left ear. A high fever was present on the following day and persisted until his admission to the hospital. Two days previously, however, he felt very much better, took a cold bath, was seemingly refreshed, but toward evening became extremely ill again and for the last eighteen hours had been

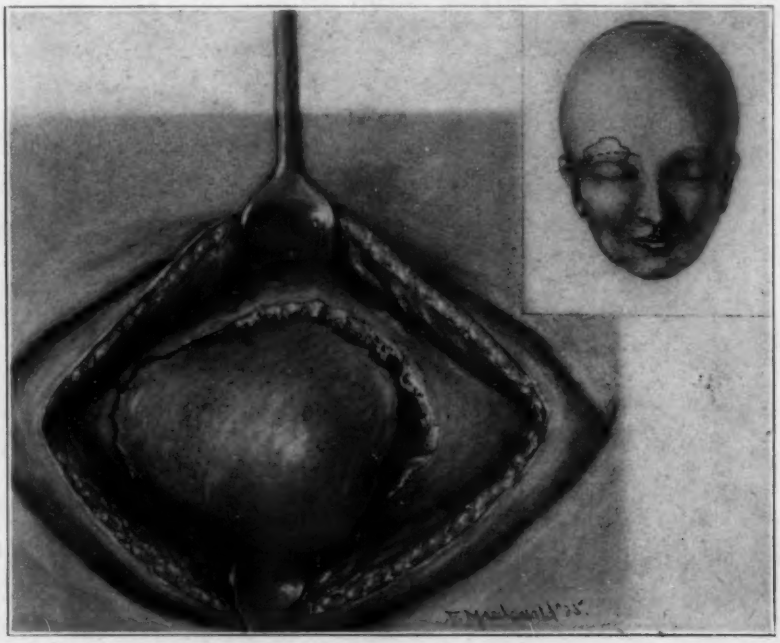

Fig. 9 (case 1).—Exposure of depressed fracture in frontal region.

delirious and unable to eat. Every day since the onset of his illness, except on the day he felt better, he had had a severe chill. His neck had been stiff throughout the week. The left mastoid was definitely tender to firm pressure. Leukocytes totaled 12,000, of which 85 per cent. were of the polymorphonuclear type. The spinal fluid was not under marked pressure (70 mm. of water). Pressure on the left jugular vein caused a rise of spinal fluid to 140 mm. and on the right to 160 mm. The spinal fluid was of ground glass appearance and contained 2,000 cells (96 per cent. polymorphonuclear). No organisms were seen in smears and none grew from cultures.

Examination of the eye grounds showed a slight but definite fulness of the veins; the disk outlines were sharply defined. The paranasal sinuses were not infected. The left ear drum was punctured but pus was not found. Owing

to the tenderness over the left mastoid in association with the sharply localized pain in the same region and a history of otitis media a year before (side not known), it seemed most probable that an old mastoid infection had eroded the bone and produced a subdural abscess, possibly invading the brain. The meningeal changes were interpreted to be "reactive" to a contiguous infection which might be extradural or intracerebral or both.

The mastoid cells were opened by Dr. Chisolm and were not found abnormal until the antrum was reached. Here a collection of pus with extremely foul odor was encountered; it was well walled off from the rest of the mastoid but it had broken through the inner bony wall and communicated with an extradural abscess. From the pus three organisms were grown: (1) a gram-negative rod bacteria, (2) gram-negative cocci and (3) diplococci. They were interpreted by the medical bacteriologic division of the hospital to be (1) *Bacillus coli,* (2) *Staphylococcus albus* and (3) pneumococcus type IV. The sigmoid sinus

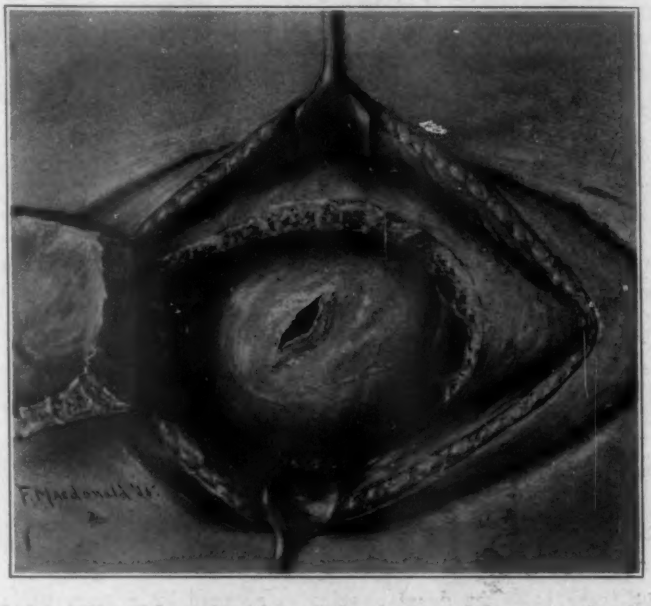

Fig. 10 (case 1).—After elevation of the depressed fracture, the dural opening was exposed behind its center; the frontal sinus was opened mesially only and it was from this source that the air was forced into the defect of the frontal lobe; it is also evident that the position of this opening formed in effect a valve that permitted the air to enter the brain but (by apposition of the dura with the bony fragment) precluded its exit.

was inspected and seemed normal and no opening in the dura was detected. The temperature fell to 99 and the pulse to 84 the following morning. However, scarcely any symptomatic improvement was apparent and forty-eight hours after the mastoid operation the temperature had returned to 104 and the pulse to 100. The previous irrational state had not changed.

An intracranial abscess seemed most likely and under local anesthesia a ventricular needle was passed through the dura into the left temporal lobe at a point directly above the mastoid. Probably 2 ounces (59.2 cc.) of a thick,

granular, greenish brown pus with a foul fecal odor and mixed with many bubbles of gas slowly escaped under great pressure. The abscess lay immediately beneath the dura. Aside from evacuation of the pus through the needle no other treatment was attempted. Since the infection was presumed to be due to a gas producing organism surgical treatment was considered futile.

When the patient was returned to the ward a roentgenogram was taken. Unfortunately one had not been taken before. The roentgenogram now showed a small oval patch (2 by 1 cm.) of air in the region of the abscess, another rounded area (7 by 1 cm.) with fringed edges just above and anterior to the first shadow, and finally a third shadow of oval shape, quite sharply outlined as though in a preformed cavity (fig. 3). It was at quite a distance from the other air shadows and suggested, both from its shape and position, the anterior horn of the lateral ventricle..

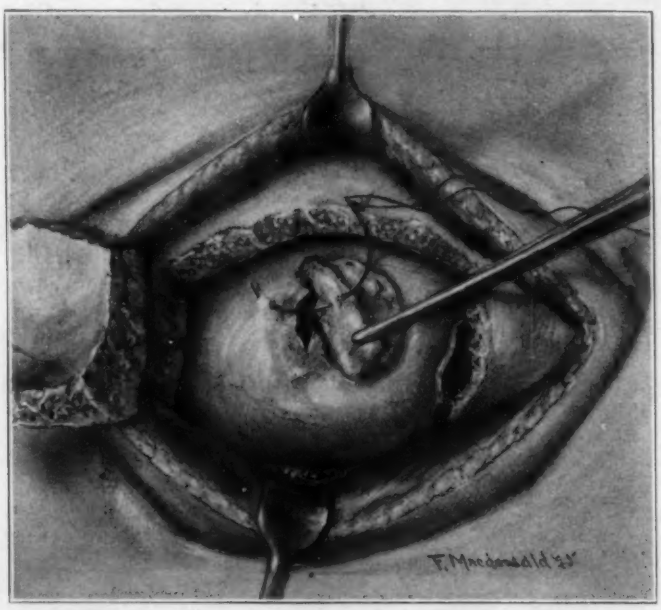

Fig. 11 (case 1).—The dural opening is covered by a piece of fascia lata, which is carefully sutured in place; the entrance of air into the cranial chamber is thus prevented.

The puncture of the abscess was followed by no signs of improvement in his condition. On the following day a second series of roentgenograms was taken. The air was still present in the anterior horn of the lateral ventricle and in the temporal lobe, but the size of the air shadows seemed unchanged. There was then reason to doubt the validity of our assumption that the air had been of bacterial origin. It seemed incredible that the volume of air should not have increased if the organisms were responsible.

To meet any question of doubt and leave nothing undone the abscess was drained. The dura was under great pressure. The abscess was entirely within the temporal lobe. From time to time pus could be seen emerging through the dura into the mastoid wound but the fistula could not be seen owing to its great depth. Again no improvement followed and three days later the patient

died. On the day before his death another skiagram showed but little difference in the air shadows—certainly the quantity was not greater. We were therefore confronted with additional evidence which seemed to be against the bacterial origin of the gas. Had bacteria been the source, there should have been an increase in the size of the air shadow. Unfortunately, the possible gas producing property of the organisms was not tested in vitro.

It is my feeling that the air was forced through the mastoid defect produced by the operation into the abscess cavity by swallowing, coughing or sneezing, just as an occipital extracranial pneumatocele has been known to form. Prior to the operation this hardly could have resulted; the abscess was well walled off and there had been no discharge from the ear. Moreover, when the cerebral abscess was drained, a periodic discharge of pus from the abscess into the

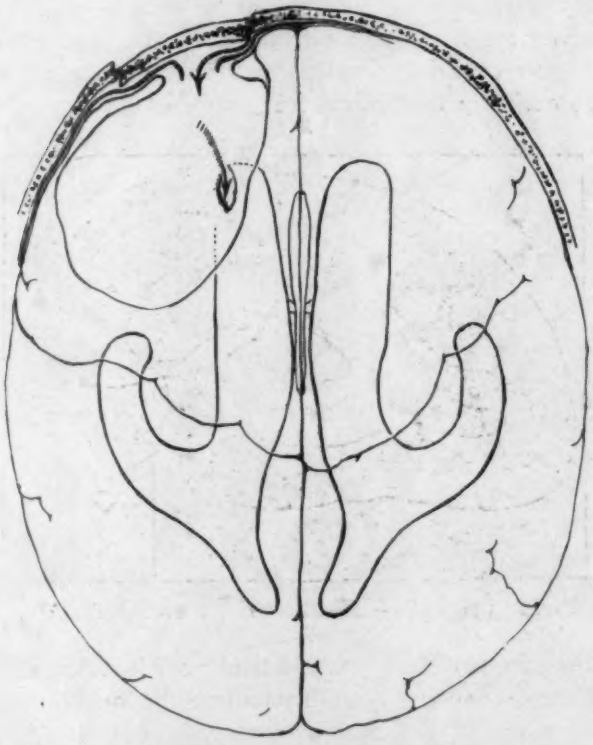

Fig. 12 (case 1).—Findings at operation; the arrows indicate the manner in which air reached the defect in the frontal lobe and by which it passed from this defect into the anterior horn of the lateral ventricle.

mastoid defect could be seen, and at that time the dura was well exposed and there was no extradural abscess.

Necropsy showed a large abscess of the left temporal lobe with a small fistula in the descending horn of the left lateral ventricle; diffuse meningitis; thrombosis of the left lateral sinus (presumably a recent development, since Queckenstedt's test on admission had indicated the sinus to be patent, though the fact that there was 20 mm. less rise of spinal fluid on compression of the left jugular vein may be an indication of a beginning thrombosis of the left lateral sinus).

CASE 3.—During a consultation with Dr. James K. Corss at Newport News, Va., I was shown a patient whose skull had been fractured a week previously. For two days following the accident, cerebrospinal fluid drained from the right ear. He had since been free of symptoms and was ready to be discharged. Being aware of the possibility of pneumocephalus as a sequel of a cerebrospinal fistula, I at once sought the roentgenograms and found air filling a lateral ventricle, the cisterna chiasmatis and interpeduncularis (in the lateral view), and both lateral ventricles in cross section in the anteroposterior view. The ventricles were small and symmetrical. Dr. Corss kindly obtained another roentgenogram; the air had then disappeared (seven days after the accident).[8]

## RÉSUMÉ OF THE LITERATURE

In Chiari's case, a chronic infection of the ethmoid cells had weakened the wall, eroded the dura and penetrated the brain. He was able clearly to show at necropsy a tiny fistulous tract leading from an ethmoid cell into a large cavity in the frontal lobe; another opening of larger size

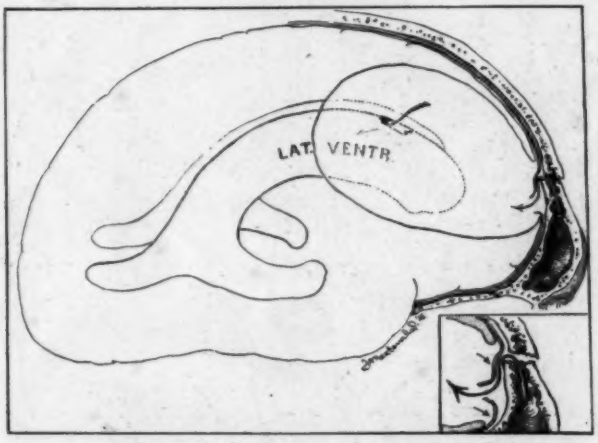


Fig. 13 (case 1).—Lateral view of operative findings.

led from this frontal defect into a lateral ventricle. Violent sneezing was one of the outstanding symptoms before the onset of severe headaches, which were soon followed by stupor and death. A diagnosis of cerebral abscess had been made. Chiari's case seems to have elicited no comment, and no other cases have been found in a search of the pathologic literature. Even at the present time textbooks either pass over the subject of cranial pneumatocele completely or casually mention only the extracranial variety.

Practically all of the cases in the German literature are due to war wounds, the infantry bullet being the usual type of injury. On the other

8. This case was found as this article was ready to be sent to press and after the accompanying figures had been arranged. As the roentgenograms would show nothing additional there seemed little point in including them.

hand, all the American cases followed injuries of civil life. Doubtless cases must have been detected in the American, French and Italian fronts during the war but no reports have been found. And doubtless also many cases have been overlooked in both civil and military life. In only one case (Wodarcz,[3] and this diagnosis was not substantiated

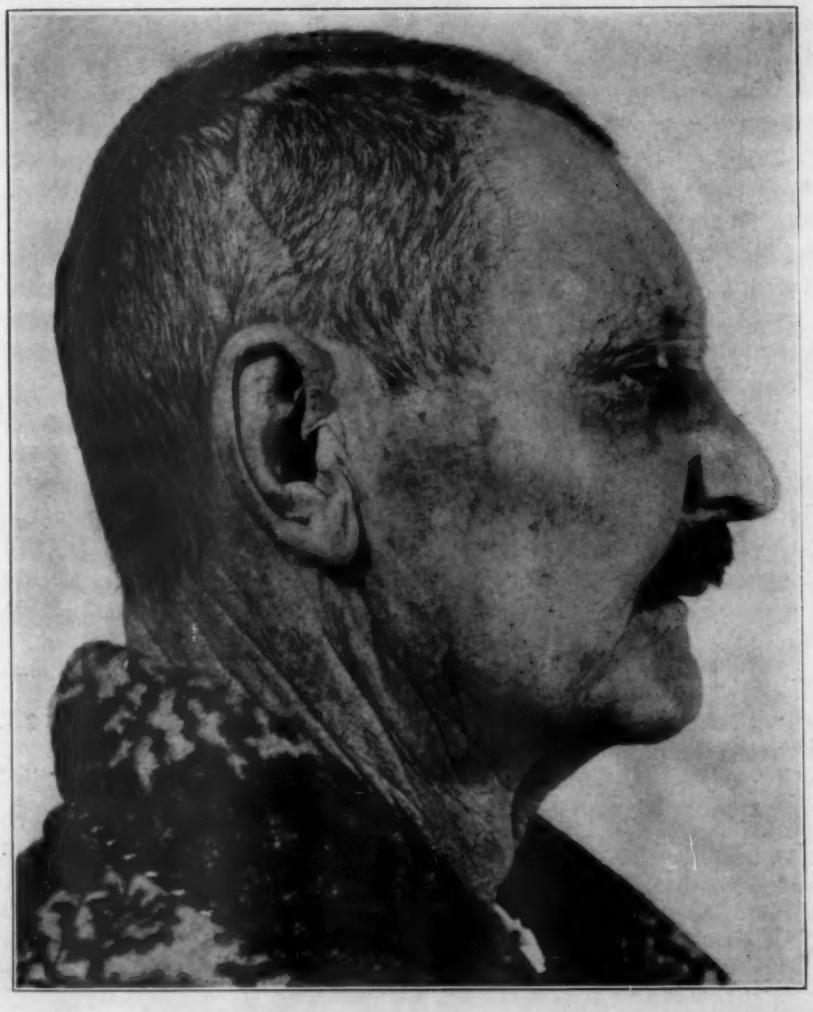

Fig. 14 (case 1).—Patient three weeks after operation; the supra-orbital scar though scarcely visible will be covered by the eyebrow; the craniotomy scar is entirely within the hair line.

by the roentgen ray) has the diagnosis of intracranial air been suspected before its discovery by the roentgen ray, operation or necropsy; usually a brain abscess or meningitis is diagnosed. An analysis of the series shows that air has entered the cranial chamber through all the cranial

air spaces, three through the ethmoids (Chiari,[2] Holmes,[9] Doyle [10] and Spiller [11]), two through the sphenoid (May [12] and Santoro [13]), four through the mastoid (Duken,[14] Teachenor,[15] Dandy,[16] two), and in the remaining cases through the frontal sinus.

Mackay [17] (1903) and Hixon [18] (1915) and doubtless others have reported instances of multiple intracranial cavities of air resulting from bacteria (presumably the *Bacillus aerogenes-capsulatus* of Welch), but the findings have been made at postmortem examinations. Since the rapidity of growth of gas from these organisms after death is well recognized, the importance of the findings of gas has been discounted. However, Hixon stresses the fact that in his case necropsy was performed only one hour after death—an interval which should surely warrant the assumption that the air had been present during life. Though gas bacillus infections were rampant during the World War and roentgen rays were employed in detecting the early traces of gas in the soft parts, I have been able to find no record in which air was found or suspected during life; but its disclosure could, of course, have been of only academic interest.

We shall consider only the entrance of intracranial air through clefts in the skull. With the exception of Chiari's case, which was of infectious origin, and Santoro's, which followed erosion of the floor of the sella turcica from local intracranial pressure (brain tumor), all have followed some form of trauma to the skull. It has been suggested that pneumocephalus is more frequent than the records indicate. Doubtless this is true and more liberal use of the roentgen ray will surely

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9. Holmes, G. W.: Intracranial Aerocele Following Fracture of the Frontal Bone, Am. J. Roentgenol. **5**:384 (Aug.) 1918.

10. Doyle, A. S.: Traumatic Pneumocranium, Am. J. Roentgenol. **8**:73 (Feb.) 1921.

11. Spiller, W. G.: Aerocele of the Brain, M. Clin. N. Am. **5**:651 (Nov.) 1921.

12. May, R. J.: Report of a Case Showing Air Within the Cranial Cavity, Am. J. Roentgenol. **6**:190 (April) 1919.

13. Santoro: Rinorrea cefalo-rachidiana e pneumoventricolo spontanei da tumore della base (ipofisi?), (Spontane Liquorrhinorrhoe und Pneumoventrikel bei einer Basis- [Hypophysen-?] Geschwulst), Riv. oto-neuro-oftalmol. **1**:484, 1924 (original not seen).

14. Duken: Ueber zwei Fälle von intracranieller Pneumatocele nach Schussverletzung, München. med. Wchnschr. **62**:598, 1915.

15. Teachenor, F. R.: Pneumoventricle of the Cerebrum, Ann. Surg. **78**:561 (Nov.) 1923.

16. Dandy, W. E.: The Treatment of Staphylococcus and Streptococcus Meningitis by Continuous Drainage of the Cisterna Magna, Surg., Gynec. & Obst. **39**:760 (Dec.) 1924.

17. Mackay, M.: A Brain Containing Gas Cyst of Microbic Origin, Montreal M. J. **32**:795, 1903.

18. Hixon: The Gas Bacillus in a Brain Injury, Brit. M. J. **2**:905 (Dec. 18) 1915.

greatly increase the number of cases. But when it is considered that a number of factors must be properly combined before air can enter and remain within the cranial chamber the total number can only be a small fraction of the rapidly mounting number of injuries to the skull.

## VARIETIES OF PNEUMOCEPHALUS, THEIR CAUSES AND MODE OF FORMATION

A study of the roentgenograms of the reported cases indicates that air entering the cranial chamber from the cranial air cells may be located in: (1) the subarachnoid space, (2) the subdural space, (3) the brain and (4) the ventricles. Obviously the location of the air is determined by the normal anatomic factors at the site of the fracture, but pathologic and physiologic factors of secondary origin also play a rôle.

From the published photographs in this series it seems certain that in at least two cases, the air is confined to the subarachnoid space, the cisterna chiasmatis and the cerebral sulci. The anatomy of the cerebrospinal spaces makes it probable that the primary entry of air into the subarachnoid space could be only through the cisterna at the base of the brain and, therefore, only from the posterior ethmoid and sphenoid sinuses. In May's case the sphenoid cells and in Holmes' case probably the ethmoids were opened. Moreover, the close application of the outer layer of the arachnoid membrane to the dura in the region of the cisterna chiasmatis would almost preclude an accumulation of air between the dura and the arachnoid, particularly so when the air can so easily enter the big subarachnoid space. On the other hand a fracture through the frontal sinus would be opposite the cerebral sulci; these are so small and the arachnoid membrane is so tightly clasped to the brain that the entry of air into the subarachnoid space from this source would be almost inconceivable. Here the subdural space, though practically a mere slit under normal conditions, is potentially a space of great size. The fact that compound fractures in the region of the cisterna chiasmatis are less common than elsewhere would seem to explain the relative infrequency of the primary entrance of air into the subarachnoid space. Although cerebrospinal rhinorrhea was not mentioned in either reported instance of this variety of pneumocephalus (May's patient died in twenty-four hours), it would appear to be an almost necessary accompaniment. The danger of meningitis must also be greater in this than in other forms because infection has such easy access to the cerebrospinal spaces. It is noteworthy that the other patient (Holmes' case) who survived the initial trauma died seven days later of meningitis.

The subdural (extracerebral) accumulations of air have exclusively followed compound fractures of the frontal sinus. The roentgenograms of this variety of pneumocephalus appear to be characteristic. An

*Analysis of Cases of Pneumocephalus Reported in the Literature*

| Cases Reported by; Date; Cause | Time Air Was First Detected after Accident or Infection | Cerebro-spinal Fistula | Sneezing | Cranial Air Sinus Involved | Signs and Symptoms | Location of Air In | Operation | Result | Findings | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Chiari..... 1884 Erosion of sinus wall from chronic infection | Infection of many years' duration | + | + Severe | Ethmoid | Headache and vomiting | Brain and ventricles | None | Death | Large cavity in frontal lobe; this opens into lateral ventricle, fistula from ethmoid cell | Postmortem examination; excellent drawings of pathologic changes |
| 2. Luckett..... 1913 Fracture | 19 days | + About a cupful of fluid would immediately follow sneezing | + | Frontal | Headache; periods of confusion; melancholy; choked disks; vomiting; semicoma; Babinski right; white blood cells, 15,600 | Brain and ventricles | Decompression needle inserted into ventricle; drainage of cisterna magna for meningitis | Death 4 days later; no organisms seen in ventricular fluid; none from cultures | Diffuse meningitis; at necropsy, air escaped from trocar in ventricle | Earlier roentgenogram of head showed no air; fissure in dura; author thought probably progressive softening took place around the air |
| 3. Wolff... .... 1914 Bullet | 7 months | ..... | .. | Frontal | Four months after accident progressive hemiplegia; convulsions 3 months later | Brain (in Rolandic origin) and ventricles | Filled sinus tract with fat | Well | ..... | Author says there must be communication between the frontal sinus, the cerebral defect and the lateral ventricle |
| 4. Duken..... 1915 Bullet | 5 weeks | ..... | .. | Frontal | Headache and feeling of pressure in left forehead; suggestive unilateral choked disk; skull tympanitic | Brain | Craniotomy; brain under pressure; finger inserted into two cerebral air pockets | Well | Air-fluid level in roentgenogram; yellowish fluid in both cerebral cavities | Author thought of some explosive gas; thought valve action resulted from a flap of bone or periosteum; roentgen-ray examination showed no air three weeks after operation |
| 5. Duken..... 1915 Fracture | ..... | ..... | .. | Mastoid | ..... | Brain (in temporal lobe beneath mastoid) | None | ..... | Small bubble of air in roentgenogram | |
| 6. Gebele..... 1915 Bullet | 6 weeks | ..... | .. | Frontal | Frontal headache; no choked disk; air-fluid noise when head was moved | Brain (two shadows) | Craniotomy; serosanguineous exudate which was sterile; drained; tampon | Death from infection | ..... | Cultures of fluid at time of operation were negative |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 7. Kredel..... 1915 Bullet | 5 weeks | + | + | Frontal | Headache and pains; no bradycardia; no choked disk | Brain | Frontal sinus opened; hollow sound to tap of knife on dura; air cavity punctured | Well | Cavity size of hen's egg; fluid, serosanguineous; drainage; no tampon; back wall of frontal sinus removed | Special mention is made that there was no splashing sound, no changed percussion note, and no valve in fistula |
| 8. Wodarcz.... 1915 Shrapnel wound | 2 weeks (no roentgen-ray examination) | ..... | .. | Frontal | Rushing noise when head was bent forward first noticed 2 weeks after accident; ceased 1 month later | Brain | Exploration of wound | Well (?) | Splinter of glass in opening; he thought it acted as a valve, as in Duken's case | No roentgen-ray examination; clinical diagnosis from sound of rushing air on change of position |
| 9. Passow..... 1916 Bullet | 1 month | + Beginning at 2 months; lasted 7 weeks; reappeared 4 months later | .. | Frontal | Headaches; rhinorrhea, 40 to 140 cc. a day; rhinorrhea brought on by bending forward | Brain (egg size) | Enlarged fistula to permit free egress of air; closed opening with fat | Well 5 months after operation; no air in roentgenogram; this was three months after accident | Smooth cavity; small opening into cerebral cavity which was 10 cm. deep | Returned to regiment five months after injury; again headaches and rhinorrhea returned; larger air shadow than on first admission |
| 10. Skinner..... 1916, reported by Am. Roentgenol. Soc., 1914 Fracture | 4 weeks | .. ..... | .. | Frontal | Headache and dizziness | Brain | Trephined over air shadow; air pocket punctured | Death from meningitis 20 days after operation | Bulging dura; air and fluid in cavity; collapse of dura after tapping | Air analyzed to eliminate other gas since a gas well was nearby; analysis showed 98.2 nitrogen, 1.8 oxygen |
| 11. Brüning.... 1917 Bullet | 53 days | ..... | .. | Frontal | Headache and dizziness; no other signs of pressure | Brain (shadow size of a goose egg) | Small bone-flap; opening enlarged; tampon and drain; removed bone to fill in defect | Well 18 months later; 1 convulsion since, but back in army | Smooth cavity | Diagnosis of abscess made; bullet entered on left and operation was on right; 14 days after operation there was more air in the roentgen-ray shadow than before operation |
| 12. Cotte..... 1917 Kicked by horse | 10 months | + | .. | Frontal | Headache | Subdural space (extracerebral) (probably) | Bone flap; did not open dura; large hernia formed at site of bone defect | Temporarily well; ultimate result not stated | Hollow sound on tapping dura with instrument | Long duration of intracranial air is noteworthy; it would appear to be extracerebral |
| 13. Reisinger... 1918 Bullet | 5 weeks | + 18 days | .. | ..... | ..... | Brain (shadow seems intracerebral but author insists air was extracerebral) | Large bone-flap; extradural gauze packs | Well | Dura lying on folds on sunken cavity after puncture; cavity not exposed | Looked on as cyst from the roentgenogram; dura not opened |

*Analysis of Cases of Pneumocephalus Reported in the Literature—(Continued)*

| Cases Reported by; Date; Cause | Time Air Was First Detected after Accident or Infection | Cerebrospinal Fistula | Sneezing | Cranial Air Sinus Involved | Signs and Symptoms | Location of Air In | Operation | Result | Findings | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|
| 14. Holmes..... 1918 Fracture | At once after accident | ..... | .. | Ethmoid ? Frontal ? | ..... | Subarachnoid space (cisterna chiasmatis) | Wall of sinus removed | Death from meningitis one week later | | |
| 15. Manasse.... Bullet | 5 weeks | ..... | .. | Frontal | ..... | Brain | Posterior wall of frontal sinus removed | Well | Cavity has smooth wall and contains fine vessels | Communication with frontal sinus not disclosed |
| 16. Glenard and Aimard 1919 Bullet | 2 months | + | .. | Frontal | Headache on left side, principally noise and bruit in head when moved | Brain | None | Died of pneumonia 6 months later | Necropsy; cavity contained 25 cc. of yellowish fluid; cavity largely filled by granulation tissue | Air present also at four and a half months, but absent at five months |
| 17. May..... 1919 Fracture | 1 day | ..... | .. | Sphenoid | Death in twenty-four hours | Subarachnoid space (cisterna chiasmatis), subdural space (extracerebral) | None | Death from cranial injury | Postmortem roentgenogram | Brief note; air in cerebral sulci and anterior to frontal lobe |
| 18. Potter..... 1919 Fracture | 1 week | ..... | .. | Frontal | Slight dizziness and slight disturbance with change of position; no more symptoms while air was increasing | Brain (egg size) and ventricles | None | Well (?); spontaneous cure | ..... | Progressive increase in size of air shadow until fifth week when the lateral ventricle was reached; progressive decrease thereafter |
| 19. Doyle..... (also reported by Spiller) 1921 Fracture | 3 weeks | ..... | .. | Ethmoid | Headache; memory defective; convulsions; irrational | Brain | Dura opened | Died next day | Air hissed out when dura was punctured at operation; probe passed through cribriform plate at necropsy | Patient was unconscious three days after accident; for necropsy report comparison should be made with Spiller's article |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 20. Horrax..... 1921 Compound fracture | Accidentally discovered 3 months after accident | ..... | .. | Frontal | No symptoms | Brain | None | Well | ..... | Lobulated shadow 1 month after accident, but this had disappeared 2 months later when patient sought relief for direct effects of injury, i. e., blindness and deafness on right; weakness of right side of face; loss of smell; strabismus and some mental aberration |
| 21. Teachenor.. 1923 Fracture | Same day | + | .. | Mastoid | Dizziness; confusion; bilateral Babinski | Subarachnoid space, subdural space (extracerebral), ventricle | None | Well | Roentgenograms show both lateral ventricles to be well filled with air | Air disappeared in 18 days; author thought air entered the floor of third ventricle |
| 22. Grant..... 1923 Compound fracture | 2 days | ..... | .. | Frontal | Confusion; restlessness | Subdural space (extracerebral) (?) (elliptical shadow) | Bone flap; nothing seen; pack left to control severe bleedings | Slow recovery | Air escaped after tight dura was tapped | Bone flap was undoubtedly made too high to disclose fistula |
| 23. McCannel.. 1923 Fracture | 4 weeks | ..... | .. | Frontal | Double vision; congested disks; slight exophthalmos; visual loss though largely the result of the fracture | Brain | None | Well; steady improvement after accident | ..... | Air was not present eight days after accident (roentgen-ray examination); in less than two more months air gone |
| 24. Imboden... Fracture | 1 month | ..... | .. | Frontal | ..... | ..... | Air removed | | | |
| 25. Santoro.... 1924 | Following erosion of floor of skull by a tumor | ..... | .. | ..... | ..... | Ventricle | None | Death from neurositis | ..... | Original article not obtainable |
| 26. Dandy..... 1925 Fracture | 6 weeks | ..... | + | Frontal | Stupor; delirium; bradycardia | Brain and ventricle | Closure of dural opening with a facial transplant | Well | Air cavity filled right frontal lobe and communicated with right lateral ventricle | Potential valve due to dura defect behind depressed fracture; this admitted air and prevented its escape |
| 27. Dandy..... 1925 Operation on mastoid | 1 day | ..... | 0 | Mastoid | Coma; high fever | Brain and ventricle | Puncture of cerebral abscess | Death from cerebral abscess | Abscess contained colon bacilli and micrococci | Air was formed in abscess and ventricle 3 days after mastoid operation; a possibility remains that the air may be of microbic origin |
| 28. Dandy..... 1925 Fracture | 1 day | + From right ear, lasted 48 hours | .. | Mastoid | Slight headache | Subarachnoid space and ventricle | None | Well | ..... | Cerebrospinal fistula lasted 2 days |

elliptical shadow, conforming in front to the curve of the frontal bone and behind to the curved surface of the frontal lobe which had been pushed backward, defines the shape of the roentgen-ray shadow. It would seem probable that the cases of Cotte,[19] of Grant[20] and of Imboden[21] are of this variety. Reisinger[22] insists on the extracerebral location of air in his case but the round air shadow (characteristic of intracerebral air) makes his claim none too certain, particularly as he did not open the dura at operation.

The intracerebral variety appears to be most common. Usually a shadow of roughly round or oval shape and of varying size projects backward from the frontal bone, no longer conforming to its contour as does the extracerebral (subdural) form. This defect often fills the entire frontal lobe and extends backward into the parietal lobe. The cavity always communicates with the frontal or ethmoid sinus by a small opening, often so minute as to be easily overlooked. In our case and so far as it is possible to judge in those reported, the dura was tightly adherent to the brain surrounding the fistula. Were it otherwise there would necessarily be a subdural collection of air in the roentgenogram; this has been uniformly absent. An intracerebral enclosure of air must therefore arise relatively late, i. e., after the traumatized brain has become adherent to the dura. It would also seem that a rupture of the cerebral cortex at the time of the accident must be a prerequisite to the formation of an intracerebral pneumatocele. Otherwise adhesions would be unlikely and entrance of the brain tissue would be difficult. The anatomy of this region is favorable to the production of an intracerebral pneumatocele, first, because the dura is so closely applied to the frontal bone that a fracture also readily tears the dura; and second, because the tip of the frontal lobe is in such close apposition to the dura that the cerebral cortex as well as the meninges are easily ruptured. But even so, it is doubtful if an intracerebral pneumatocele or the other forms of pneumocephalus could develop without an added factor, i. e., the increased pressure of the air within the cranial sinuses such as is produced by sneezing, coughing, straining or possibly even of swallowing.

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19. Cotte, G.: Hydropneumatocele traumatique du crâne, Bull. et mém. Soc. de chir. de Paris **43**:865-867, 1917.

20. Grant, F. C.: Intracranial Aerocele Following a Fracture of the Skull: Report of a Case with a Review of the Literature, Surg., Gynec. & Obst. **36**:251 (Feb.) 1923.

21. Imboden: American Atlas of Stereoroentgenology **11**, cited by McCannel, A. D.: Aerocele of the Brain with Report of Cases, Laryngoscope **33**:189 (March) 1923.

22. Reisinger: Ueber intracranielle, aber extracerebrale Pneumatocele nach Schussverletzungen, Beitr. z. klin. Chir. **109**:129, 1918.

The small size of the sinus tract between the pneumatocele and the sinus has often been commented on; in fact, at operation an opening has not always been found. Moreover, the fact that intracranial pressure has developed is sufficient evidence that a compensatory escape of fluid or air or of both from the cranial chamber is not possible. Because of these facts there has been speculation about a probable valve action in the fistula opening. Wodarcz found a fragment of glass in the communicating tract and considered this to act as a valve. Duken could find no definite valve but thought a piece of granulation tissue or bone must have served this function. Kredel [23] could find no valve and considered a valve unnecessary if the fistula were small. In our case there was a potential valve in that the dural opening was behind the center of the depressed fragment of bone and not, therefore, in a direct line with the fracture. Sneezing would push the dura away from the bone and readily force the air through the relatively large opening of the dura into the cerebral defect; after this the heightened intracranial pressure would seal the dura against the depressed bone and effectively close the opening. But we have every reason to believe that an actual valve is not necessary for the induction of pneumocephalus even when the fistulous tract may be perfectly straight. If the fistula is of just the right size (both length and breadth) and its walls not too rigid it would naturally be widened by the explosive force of sneezing and permit the quick passage of air into the cranial chamber, but the vis a tergo of the lesser and steadier pressure within the cranial chamber, even though above normal, would not be able again to distend the opening and allow the air to escape from the cerebral defect. An opening of an ideal size might also well permit the escape of fluid—cerebrospinal rhinorrhea—and yet prevent the egress of air. The results of three tests may be cited as experiments to prove these statements:

1. An opening may be of such size that fluid can and air cannot pass. Indigo-carmine and air were injected into the *right* lateral ventricle of a patient suffering from the effects of a brain tumor that was found to be located in the third ventricle. The dye was at once and freely aspirated through a needle in the *left* lateral ventricle. But the air which had been injected into the right lateral ventricle at the same time failed to reach the left lateral ventricle. The absence of air in the left ventricle was shown not only by the failure of air to be aspirated but also by the absence of an air shadow in the left side of the head, though the right was well filled.

2. Additional evidence that a channel may be patent for fluid and not for air has been included in a recent paper on the diagnosis and

23. Kredel, L.: Die Intrazerebrale Pneumatokele nach Schussverletzungen, Zentralbl. f. Chir. **42**:649, 1915.

localization of spinal cord tumors.[24] It was demonstrated that fluid will often pass freely around a spinal cord tumor (Queckenstedt's test) when air will stop at the lower level of the tumor.

3. Finally, continuing further the test just mentioned, if the intraspinous pressure is increased, the air may be forced beyond the spinal cord tumor which under normal pressure had indicated a complete block of the subarachnoid space. An example of this kind has been reported under the classification of partial obstructions in the same paper on spinal cord tumors. The factors involved in this experiment are precisely the same as those in the development of pneumocephalus.

But the development of an intracerebral pneumatocele is the result of more than the mechanical entrance of air into the brain. Definite pathologic reactions follow the entrance of air and these play a big rôle in the development of the end product as we see it at operation or necropsy. From our experience with cerebral pneumography we know that air is an irritant. After the initial traumatic insult which results to the brain tissue from the explosive entrance of air there is a secondary reaction due to the *irritation* of the air, not unlike, except in degree, the reaction which would follow the introduction of any irritating liquid. One result of the irritation is a serosanguineous fluid which, pouring into the cerebral defect, produces additional local as well as general intracranial pressure. Another result is an edema of the brain tissue contiguous to the defect and resulting both from the traumatic insult and the subsequent irritation of the air. Although life may still be tolerated the loss of much brain tissue must inevitably follow in the wake of these changes. In Luckett's [3] early case it was assumed that a gradual softening of the brain tissue resulted from the effects of the air. We have reasons to believe that the white matter of the brain is much less resistant to trauma than the cortex. The cortex is actually harder to cut; pressure of a spatula quickly produces petechial hemorrhages and edema in the exposed white matter though the same insults will be tolerated by the cortex and its protecting meninges. The white matter, therefore, presumably suffers first and most in the cerebral absorption resulting from pressure and irritation of the air in pneumocephalus.

In many of the recorded cases of intracerebral pneumocephalus a cerebrospinal rhinorrhea has been reported. As long as the cerebral defect is not in continuity with either the ventricles or the subarachnoid space the fluid that escapes must be a serosanguineous reaction product. In some instances (Duken,[14] Gebele [25]) a fluid-air level has been dis-

24. Dandy, W. E.: The Diagnosis and Localization of Spinal Cord Tumors, Ann. Surg. **81**:223 (Jan.) 1925.

25. Gebele: Ueber Schussverletzungen Des Gehirns, Beitr. z. klin. Chir. **97**: 123-145, 1915.

closed in the roentgenogram and in one case [26] there is the characteristic story that fluid pours from the nose when the patient bends forward, i. e., when the dependent fluid can pour over the brim of the cerebral cavity as from a cup. A single instance of this kind demonstrates that a valve is neither present nor necessary for the development of pneumocephalus. Duken [14] found a yellow, Kredel,[23] a serosanguineous, and Glenard and Aimard,[27] a lemon yellow fluid. Though there is no evidence contained in this series of cases it seems safe to reason from the known facts that the rhinorrhea of an intracerebral pneumatocele must be less in amount and different in character from that of pneumocephalus of the subarachnoid or intraventricular types in which pure cerebrospinal fluid can escape freely.

Intraventricular pneumocephalus is often but a later stage in the progress of an intracerebral pneumatocele, though the air may enter directly into the ventricles from the subarachnoid space when this variety of pneumocephalus exists. It has been repeatedly shown that air injected into the subarachnoid space through the spinal canal at once passes into the ventricular system. Teachenor's case is an interesting example of the rapidity with which air may reach the lateral ventricles—a few hours. Although Teachenor makes the interesting assumption that the air might have entered the ventricles through a tear in the floor of the third ventricle, I should feel more inclined to believe it entered through the normal channels—the foramina of Luschka and Magendie. This will always be expected when the air is in sufficient quantity in the subarachnoid space. Our case (case 3) is practically a duplicate of Teachenor's.

In the metamorphosis of the intracerebral pneumatocele the frontal defect gradually enlarges and eventually opens into the lateral ventricle of the corresponding size. In our case, and apparently also in Chiari's, the ventricular opening was about as large as the circumference of a slate pencil. Once in the ventricle, the air has free access to every part of the ventricular system and emerges into the subarachoid space. Precisely as in deliberate cerebral pneumography for diagnostic purposes every part of the ventricular system can then be clearly shown in the roentgenograms.

We also know from experiences with cerebral pneumography that air absorbs almost entirely in the subarachnoid space. It is probable, therefore, that coincident with the rupture of air into the lateral ventricle there may be a transient period of relief from symptoms, i. e., until the cranial chamber is again overwhelmed by a new supply of air from the

26. Passow, A.: Ueber Luftansammlungen im Schädelinnern, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **8**:257, 1916.

27. Glenard and Aimard: Aérocèle traumatique du cerveau, Presse méd. **27**:123 (March 10) 1919.

nasal sinuses. We were interested to pick out if possible any such period in our patient. Fifteen days before his arrival in Baltimore he rather suddenly and surprisingly came out of a deep stupor. A roentgenogram was not taken, however, until six days later; at that time the ventricles contained air.

### SYMPTOMS OF PNEUMOCEPHALUS

Occasionally the symptoms are masked by the greater local disturbances of the fracture as in Horrax's case.[28] Teachenor's patient had surprisingly mild symptoms, doubtless because the fistula through which the air entered was soon closed. On the whole the symptoms of any form of pneumocephalus are severe. With few exceptions they are those of intracranial pressure, headache, vomiting, drowsiness, possibly double vision and dizziness, and finally restlessness, delirium and coma. Papilloedema has been noted in a few cases by Luckett,[3] McCannel,[29] and Duken.[14] It has been noted as absent in others by Gebele,[25] Kredel,[23] Teachenor,[15] and in all of our cases.

It hardly seems possible to make an absolute diagnosis of pneumocephalus from any combination of signs or symptoms except one, sneezing followed by cerebrospinal rhinorrhea. One usually can only suspect this condition and confirm or disprove the suspicion by the pathognomonic roentgen-ray findings. All fractures involving the sinuses should place one on guard for this diagnosis, whether the symptoms of intracranial pressure follow immediately or, as so often happens, at a latter date. A cerebrospinal fistula is probably the sign of greatest significance. Its presence should always make one suspect pneumocephalus. It was solely on the basis of this manifestation that air was suspected and found in our third case. When sneezing follows a frontal fracture with rhinorrhea, the suspicion grows stronger, but when sneezing is followed by a flow of cerebrospinal fluid, as in Luckett's case, one could almost be safe in making a positive diagnosis of pneumocephalus without the roentgen ray. There are practically only four conditions which can appear as late (from several days to weeks after injury) sequelae of a fracture, subdural venous hemorrhage (erroneously called pachymeningitis haemorrhagica), brain abscess meningitis and pneumocephalus. As said before the roentgen ray at once makes or eliminates the diagnosis of air in the cranial chamber, thus making the differential diagnosis easy.

In Wolff's [4] patient hemiplegia gradually developed and convulsions followed; Doyle's and Spiller's patients also had convulsions; Luckett's patient had a unilateral positive Babinski, Teachenor's bilateral. Other

28. Horrax, G.: Intracranial Aerocele Following Fracture of Skull, Ann. Surg. **73**:18 (Jan.) 1921.

29. McCannel (Footnote 21).

patients have had defects of vision and smell, ptosis, strabismus, etc., all, however, caused by cerebral injury due to the initial trauma and not related to the pneumocephalus. As mentioned before, only one case was suspected from clinical symptoms. In our third case, air was suspected, not from symptoms, but because of the presence of a cerebrospinal fistula. Among the earlier reported cases even the interpretation of the roentgen-ray shadow was most uncertain. Skinner [30] (1916), for instance, thought of the possibility of some gas from nearby gas wells and had it analyzed only to find ordinary air. Duken [14] (1915) thought of some explosive gas, and Brüning [31] looked on the shadow as that of an abscess. Almost without exception headache has been the outstanding complaint and this symptom together with stupor was the combination of symptoms referable to the brain.

In three cases, those of Holmes, May and Teachenor, air was present in the cranial chamber when roentgenograms were taken a few hours after the accident and in Grant's case and in Case 3 of this report it was present in the first roentgenogram, which was taken on the second day. The time at which the air was detected in the remaining cases is doubtless always considerably later than the actual appearance time. It is fair to assume that in some instances it must have been present at least soon after the accident and had steadily increased until the cerebral lesion was large enough to cause intracranial pressure. In McCannel's case air was absent eight days after the trauma and present at the next roentgenogram, three weeks later. In my first case air was first detected in the cerebral ventricles five weeks after the accident, and in Potter's [32] case air was in the brain during the first week and in the ventricle when another plate was taken a month later. In eleven cases of the total number the air has been recognized in less than a month after the accident, and in ten instances between the fourth and the sixth weeks. In three cases air was recognized only after a longer time, by Wolff in seven months and by Cotte in ten months. Luckett's case (nineteen days) indicates how short a time may be required for air to traverse the frontal lobe and break into a lateral ventricle. On the other hand Passow's case (eight months), Cotte's (ten months) and Brüning's (seventeen months) show how long the air may exist in the frontal lobe and yet not enter a ventricle. It seems probable from Cotte's report that the air in his case was extracerebral; if this is true it is an example in which air did not penetrate the brain even after ten months. As cerebral destruction doubtless

30. Skinner, E. H.: Intracranial Aerocele, J. Am. M. Soc. **66**:954, 1916.

31. Brüning, F.: Uebergrosse lufthaltige Gehirncyste nach Schussverletzung, Beitr. z. klin. Chir. **107**:432, 1917.

32. Potter, H. E.: A Case of Hydropneumocranium with Air in the Ventricles, Am. J. Roentgenol. **6**:12 (Jan.) 1919.

occurs rapidly both from the irritating effect of air and from the traumatic effects of the explosive force of air, it is difficult to arrive at a conclusion regarding the relative effect of each. The rapidity with which the pneumocephalus develops must also be dependent on such variable factors as the amount of sneezing, the amount of initial cerebral destruction, the size and elasticity of the fistulous tract and also on the presence or absence of a valve. A large opening without a valve is undoubtedly more favorable, other things being equal, because the outflow of fluid and air spontaneously relieves intracranial pressure. Passow's patient was cured by making the sinus larger, thereby preventing retention.

Several cases have been observed over a period of weeks and even months after the air was known to be present in the brain, some with and others without surgical treatment. One of the most interesting of these was Passow's case. One month after a bullet wound in the supra-orbital region, a clear patch was seen in the roentgenogram of the frontal lobe. Although the shadow was absent in the plate taken soon after the accident, the significance of its later appearance was not realized. Five months later the patient was apparently well and returned to his battalion and a pronounced cerebrospinal rhinorrhea, which on the earlier admission had persisted for six weeks, had then been absent two months. However, after a month's activity the same headaches again returned and two weeks later rhinorrhea followed. The air shadow was larger than before. He was then operated on and after another five months, or thirteen months after the accident, the air had disappeared. Potter watched the shadow of air increase in his case over a period of a month; the air finally reached the lateral ventricle after which it soon disappeared.

In Teachenor's case a great quantity of ventricular air disappeared in eighteen days. The importance of a cerebrospinal fistula as a diagnostic sign cannot be overemphasized. It has been recorded in only nine cases though doubtless it has been overlooked in many, owing to the failure to suspect the diagnosis of pneumocephalus. Overlooked in one of our cases at the time the history was taken, it was later found to be an outstanding and distressing symptom. In Luckett's case about a cupful of clear fluid suddenly *followed sneezing,* the fluid probably being released as the air was driven into the cerebral cavity. Passow's patient drained continuously for two months, at first 140 cc. per day; gradually the flow lessened to 40 cc. per day and ceased, later reappearing after a free interval of four months. Though a history of rhinorrhea could not be elicited in our case, it is difficult to believe it was absent, even with the undoubted valve action in the fistula. Sneezing was recorded and emphasized in the early reports of Chiari and of Luckett, and later of Kredel. It is doubtless the great active factor in the development of this condition.

An instance of rhinorrhea with change of position has been mentioned (Passow's case). This can, of course, appear only in those cases in which the fistula is relatively large and no valve is present. Such an observation, when questioned, should be almost as significant as rhinorrhea following a sneeze. At times the patient heard the gurgling sound in his head on turning, an observation now frequently made in deliberate injection of air into the ventricles. Duken mentions a tympanitic note over the air space, a sign that Kredel was unable to elicit. Wodarcz's patient heard a rushing sound when he moved his head and on this symptom, the diagnosis of pneumatocele was made. This characteristic noise is well known to patients in whom a deliberate injection of air has been made into the ventricles.

### THE PROGNOSIS

Excluding May's case, in which death soon followed the accident and was in no way dependent on the pneumocephalus, there are sixteen recoveries and eleven deaths. Of the sixteen recoveries, seven were entirely spontaneous and nine followed operative procedures of various kinds. Undoubtedly several of the postoperative recoveries would have occurred spontaneously for the operation accomplished nothing more than the discovery of the lesion. It is also fair to add that many of the postoperative deaths would have occurred had nothing been done. On the whole the mortality would probably average about 50 per cent if the patients were left untreated. The deaths are due to two factors: (1) intracranial pressure and (2) infection from a compound wound. That air can pass through a paranasal or mastoid air sinus into the brain—even into the cerebrospinal fluid—over a period of weeks and months without infection speaks well for the sterility of the paranasal —principally frontal—sinuses in these cases. But the frequency of meningitis (six cases) and abscess (one case) attests the fact that the sterility of the sinuses is purely a matter of chance. Once an infection develops there is only a bare possibility of spontaneous recovery and a greatly reduced chance from operative treatment.

Nature cures pneumocephalus by closing the fistula with granulation tissue after which the air within the brain absorbs, slowly in the cerebral cavity, much faster from the ventricular system and subarachnoid space. Doubtless some fistulas are prevented from closing by the inclusion of a small foreign body, such as a piece of glass or bone, others because periodic sneezing overcomes the tissue growth, and doubtless others because of infection. It is worthy of note that only in Potter's case [32] has a spontaneous cure resulted after air has reached the ventricle after transit through the frontal lobe. The entrance of air into the ventricle must have been well timed with the closure of the external fistula. When the fistula has finally closed, the cerebral defect

remains filled with ventricular fluid if there is a communication with the ventricle; or at first with fluid and ultimately with granulation tissue if the cavity is isolated from the cerebrospinal spaces. The report of Glenard and Aimard shows the result of healing in such a cavity six months after the air shadow had disappeared and the patient was free of symptoms.

### OPERATIVE TREATMENT

The attempts to cure this condition have been most varied. In several instances in which a flap has been turned down and the air evacuated after being accidently found, the communicating fistula has not been treated and the patient has obtained no benefit. Brüning [31] Kredel,[23] Manasse [33] and Passow [26] have removed the posterior wall of the frontal sinus and apparently some of the frontal bone above; they then produced and maintained a large opening between the cerebral cavity and the exterior, the object being to prevent retention of air by providing a large exit. All of these patients recovered. It should be noted, however, that in none was there air in the ventricles. It is hardly possible that any would have recovered with this treatment had the air been in the ventricles at the time of operation for not only would the air become trapped in the ventricles but a cerebrospinal fistula of such size and under these conditions could hardly exist without the development of meningitis. It is of interest that fourteen days after operation Brüning found the air shadow to be larger than before operation. Following a procedure of this kind the cerebral defect must heal from the bottom, filling with granulation tissue. In Passow's case this required five months and in Brüning's case a small shadow still existed fifteen months later. In all of these cases, therefore, the treatment has been directed toward the effect rather than the cause. In addition to producing a large vent for the air cavity, Passow introduced a piece of fat into the fistulous tract hoping to impede the inflow of air from the frontal sinus, but the fate of the fat is not recorded.

It must be remembered that much of the attempted surgical treatment for this condition has been evolved on the spur of the moment and at a time when the underlying pathology was little understood. With an appreciation of the cause there can be little uncertainty in most cases as to the character of the treatment or hesitancy as to its application, namely, in the correction of the cause, i. e., closure of the fistula if it is accessible. This should surely be done as quickly as possible after the character of the lesion has been recognized. The mortality resulting when the fistula is left to the chance of spontaneous cure is too great. The risk of the suggested procedure should be little if anything. Needless to add every effort must be made to learn beforehand the exact

33. Manasse, cited by Frenzel, K.: Die Pneumatocele des Schädels, Inaugural Dissertation, Breslau, 1919.

location of the fracture, for the dural opening must be directly beneath or at least close by. In depressed fractures of the frontal bone—the most common form—the history, examination and study of the stereoscopic roentgenograms (both lateral and anteroposterior views) should make the site of the opening certain and it should be easy of operative approach.

An operative treatment is here suggested for those cases in which the fistula results from a fracture of the frontal sinus. These are easy of approach and seem to be the most frequent type. The procedure is that which was carried out in our first case. Although this treatment is not proposed for communications into the ethmoid and mastoid sinuses in which the demonstration of the opening by operation will be more difficult, it is not improbable that in some of these, at least, closure of the dural opening in a similar manner may yet be practical.

After the intracranial pressure is relieved by a ventricular puncture (if there is air in the ventricles) or by tapping the pneumatocele (whether intracerebral or extracerebral), a small incision is made along the line of the shaved eyebrow, the depressed fragment is elevated, and the opening in the dura is located and covered by a transplant of fascia lata which is carefully sutured to the dura. In our case this was particularly easy because of the isolated depressed fracture. Should there be only a crack in the posterior wall or the fragment small and impossible of ready elevation a small bone flap will give ample exposure to permit careful inspection of the dura and suture of the fascial graft. Unless infected, air already in the brain will be taken care of automatically by absorption. This plan of attack should be effective for air accumulations whether extracerebral or within the frontal lobe or, as in our case, within the ventricle. Since the operation is entirely extradural there is no danger of introducing an infection within the cranial chamber. Should a cerebral abscess be present at the time, it should, of course, be handled precisely as an abscess of other origin. If there is meningitis the best chances would appear to follow closure of the fistula, whereby the source of more infection is prevented, and after this, continuous drainage of the cisterna magna through a small catheter.

## SUMMARY AND CONCLUSIONS

1. Pneumocephalus may follow in the wake of any artificial communication between the paranasal or mastoid sinuses and the cranial chamber. The opening may result from a fracture, from an operative defect, from the erosion of a chronic infection, from destruction of the floor of the skull by a tumor or a dilated third ventricle in hydrocephalus. Pneumocephalus also results directly from infections of gas producing organisms—*Bacillus aerogenes-capsulatus* (of Welch) and possibly colon bacillus.

2. Twenty-eight cases of pneumocephalus, including three of our own, have been compiled from the literature and analyzed.

3. Sneezing, coughing, straining or swallowing is necessary to force the air through a bony and dural defect into the cranial chamber.

4. Intracranial air may be subdivided into four varieties according to location: (1) subarachnoid, (2) subdural, (3) intracerebral and (4) intraventricular. The intracerebral variety appears to be the most frequent.

5. The source of air may be the frontal, ethmoid, sphenoid and mastoid sinuses. The variety of pneumocephalus is dependent on the sinus involved, the exact character of the fracture and the duration of the air in the cranial chamber.

6. The symptoms are mainly those of increased intracranial pressure. Frequently symptoms follow weeks or even months after a trivial injury of the head. A discharge of cerebrospinal fluid is usually present; its presence should always make one suspect pneumocephalus. Sneezing is a frequent symptom. Rhinorrhea after sneezing or after change of position is almost pathognomonic of this condition.

7. The absolute diagnosis is made by the roentgen ray.

8. When left to chance the mortality of pneumocephalus is around 40 per cent., infection and pressure being the causes of death. For fistulas through the frontal sinuses an operative treatment is proposed. The dural tear is located and covered by a transplant of fascia lata, which is carefully sutured in place.

# THE TREATMENT OF OLD CONGENITAL DISLOCATION OF THE HIP

## WITH SPECIAL REFERENCE TO THE USE OF SKELETAL TRACTION BEFORE REDUCTION BY OPERATION *

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In the majority of cases of congenital dislocation of the hip in young children, reduction can be secured by manipulation. If the head of the femur is replaced without trauma and the after-care is skilfully given, good functional results are frequently obtained. In children more than 7 years of age, however, the prospect of cure by manipulation is much less bright, and in those who have reached the age of 10 or more, the pathologic changes in and about the hip joint have become so marked that reduction by this method is exceedingly difficult and frequently impossible. Furthermore, in these older children the so-called "bloodless method" is often applied with considerable force, which not uncommonly causes serious injury of the sciatic nerve, rupture of blood vessels or even fractures of the femur. Attempts at reduction by operation are often disappointing, for the surgeon will generally find that even after extensive division of the soft parts the head of the femur cannot be brought down to the level of the acetabulum. In those cases in which the disability is marked, resection of the head and neck of the femur with arthrodesis of the trochanter to the acetabulum may improve the stability of the joint, but the real value of such a procedure may be questioned because it leaves the patient with a considerable and permanent shortening of the limb.

The results obtained, therefore, by the foregoing methods leave much to be desired, and it is not surprising that many surgeons pronounce these old congenital dislocations of the hip as incurable rather than subject the patients to treatment that involves great risk and at the same time offers so little hope for cure. While this conservatism is commendable in a small group of dislocations in which stability is good and shortening only moderate, yet it is to be condemned in a large group in which there is instability of the joint, marked shortening of the limb and a well developed lumbar lordosis. It is in the latter group that we find symptoms which, in later life, lead to serious disability and not infrequently render the patients incapable of pursuing the ordinary occupations. Moreover, palliative treatment in adults often fails to give relief, and it was my own failure to obtain improvement in several of

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these patients which led me to a further study of congenital dislocation of the hip in older children with the hope that a method could be developed by which reduction could be effected. It was not expected that complete functional cures could be established by reduction; but it did not seem unreasonble to assume that the increased stability of the joint and the greater length of the leg obtained by replacement and retention of the head within the true acetabulum would give great improvement and prevent the severe disability that is so commonly seen in the untreated cases.

This study has given me a much better understanding of the pathologic changes that are the direct cause of failure to secure reduction when the usual methods of treatment are employed. The most important change is a structural shortening of the soft parts, especially of the long muscles of the thigh, which fixes the head of the femur in its position well above and behind the acetabulum. Next in importance is the change in the acetabulum, for this structure is usually undeveloped because of lack of contact of the femoral head, and in place of a socket of normal depth there remains only a shallow cavity filled with fat and folds of capsule, the latter not infrequently being adherent to its brim. With the recognition of these pathologic changes as the chief obstacles to reduction, the indications for treatment become quite clear. The first indication is to overcome the structural shortening of the soft parts so that the head of the femur can be brought to the level of the acetabulum, while the second indication is to clear the acetabulum of its contents so that the head of the femur can be placed within it. The first indication is met by the application of efficient traction; the second by operation.

The importance of efficient traction cannot be overestimated because the head of the femur must be brought directly opposite the acetabulum before any assurance can be given that reduction can be accomplished by operation. It is necessary, therefore, that the most efficient type of traction be employed. There are only two forms from which to choose, the indirect as obtained by adhesive tape applied to the skin with weight and pulley attached (Buck's extension) and the direct or skeletal, in which the pull is taken on the bone. The former, skin traction, I have found to be totally inadequate, even when used with weight up to 40 pounds. Furthermore, it is irritating and often slips, tearing off large areas of skin, which makes its reapplication impossible, to say nothing of the discomfort to the patient. On the contrary, skeletal traction as obtained by the Steinman pin is effective and the head of the femur can readily be brought to the level of the acetabulum (figs. 6 and 9).

In young children I prefer the Steinman pin rather than calipers because in one young girl on whom calipers were used they slipped into the epiphyseal line and caused a separation of the lower femoral

epiphysis. Since then I have used the Steinman pin placed through the shaft of the femur well above the epiphyseal line.

To make traction more effective I have secured countertraction by suspending the ring of a Thomas splint against the tuberosity of the ischium, after the method of Pearson. The advantages of this form of countertraction over that obtained by utilizing body weight are that it does not necessitate a dependent position of the head and that it is not altered by the patient's position in bed. It has proved to be a simple and effective type of countertraction (figs. 1 and 2).

Fig. 1.—Thomas splint suspended to Morison overhead bed frame with Steinman pin traction in a case of old congenital dislocation of the hip.

It was thought that the difficulty of bringing the head of the femur downward could be lessened by fasciotomy of the flexor muscles of the hip, which have their origin from the ilium. This operation was carried out in two of the cases cited below and the origin of the tensor fascia femoris and the pelvic origin of the rectus muscle were freed subperiosteally before the Steinman pin traction was attempted. In comparison with other cases in which this treatment was not carried out there seemed to be no appreciable difference in the facility with which descent of the head was effected.

The question as to danger of infection always arises when skeletal traction is used, especially when the Steinman pin is employed. While this danger exists it can be minimized by a scrupulous attention to the

most rigid aseptic technic during the insertion of the pin and by avoiding any change of dressing until it is removed. With these precautions I have found that the danger of infection is of no practical importance.

This method of treatment by Steinman pin traction and operation can best be described under the following headings:

1. Insertion of the Steinman pin and application of Thomas splint.
2. Fixation of the splint to the overhead bed frame and application of traction.
3. Reduction by operation.
4. Postoperative care.

Fig. 2.—Closer view of apparatus shown in figure 1; the method of suspension of the leather ring to secure countertraction is clearly shown.

### 1. INSERTION OF THE STEINMAN PIN AND APPLICATION OF THOMAS SPLINT

Twenty-four hours before operation the skin of the thigh and calf is shaved, scrubbed with soap and water and covered with a sterile dressing. At operation the skin is painted with 5 per cent picric acid solution. Sterile dressings are applied. At a point on the internal surface of the femur, 1 inch (2.5 cm.) above the adductor tubercle, the skin is pulled upward and a stab incision made down to the bone. A Steinman pin in a drill chuck is passed through the incision and turned through the femur and soft parts until its point projects beneath the skin on the lateral aspect of the thigh. Again the skin is pulled upward,

incised and the pin pushed through this incision until its extremities are of equal distance from the internal and the external surfaces of the thigh. Gauze dressings saturated in 5 per cent picric acid solution are applied to the wounds and the Steinman stirrup is fixed to the ends of the pin. It is of the greatest importance to pull the skin upward when making the incisions so that when it is released it will lie free below the pin. The absence of tension on the skin is the secret of painless traction with either calipers or Steinman pin. A Thomas splint is applied, and while the ring is held firmly against the tuberosity of the ischium, flannel bands are fastened to the lateral bars of the splint for support of the limb. The patient is now transferred to the ward.

### 2. FIXATION OF SPLINT TO THE OVERHEAD BED FRAME AND APPLICATION OF TRACTION

For overhead suspension we are using a modification of the Morison bed frame designed by Robert Morison, Sanitary Engineer of the Royal Infirmary, Edinburgh (figs. 1 and 2). It is constructed of gas pipe with tubal arms attached which can be moved up and down or rotated on the uprights. This feature allows for flexion, abduction and adduction of the limb without altering the position of the patient. The frame is simple and permits of easy and quick adjustment.

To adjust the patient to the frame a rope is attached to the anterior half of the Thomas splint and the free end passed over two pulleys on the horizontal bar. To this free end sufficient weight is attached to hold the ring firmly against the tuberosity of the ischium. The advantage of such an arrangement is that when the patient raises himself from the bed the ring continues to hug the ischial tuberosity and thus a constant countertraction is maintained. The lower end of the splint is fixed to the horizontal bar and traction is maintained by a weight and pulley attached to the Steinman stirrup.

The initial weight used is about 15 pounds, but this should be increased daily up to 30 or 40 pounds. Daily measurements of the two legs should be made and recorded. When the legs are of equal length roentgenograms should be taken to verify measurements and to determine the exact position of the head of the femur in relation to the acetabulum (figs. 6 and 9). When the head of the femur has reached the level of the acetabulum it is maintained in this position by using adhesive tape traction and the Steinman pin is removed. The object of changing the method of traction at this time is to allow the wounds made by the Steinman pin to heal before reduction by operation is attempted. The time required to bring the head of the femur down to the level of the acetabulum varies considerably, but the average period is from four to five weeks.

Pressure sores over the ischium are evidence of failure to care for the leather on the ring and the skin of the thigh with which it lies in contact. This can be avoided by changing the area of the skin pressed on at frequent intervals during the first few days of traction and by carefully washing and drying the leather ring at least twice daily.

### 3. REDUCTION BY OPERATION

The Smith-Peterson incision is used because it gives a complete exposure of the joint. After separation of the tensor fascia femoris from the sartorius muscle, the dissection is carried upward along the outer border of the rectus muscle until its origin from the upper edge of the acetabulum is seen. The origin of the gluteus medius and the gluteus minimus muscles is reflected subperiosteally from the lateral surface of the ilium in a backward direction until the upper part of the acetabulum is exposed. The rectus muscle is then retracted inward and the capsule of the joint freed from adjacent structures. An incision is made on the anterior surface of the capsule beginning at the junction of the neck and the great trochanter and ending at the lower angle of the subperiosteal flap. In this way a good view of the acetabulum, the wing of the ilium and the head and neck of the femur is obtained.

Pathologic changes in the joint can now be noted and, though there is considerable variation, they are generally present as follows:

1. A shallow and broad acetabulum with absence of a well defined upper border. The cavity is often filled with fat while folds of capsule are adherent to its rim.
2. A short and stubby neck of the femur.
3. Anteversion of the neck of the femur, varying in degree.
4. Coxa vara.
5. Small atrophic head of the femur.
6. Distorted capsule with great thickening of its upper and posterior surfaces.

The most striking features of the cases in which I have operated have been the smallness of the head of the femur and the broad but shallow acetabulum. The disproportion in the size of these two structures, together with the absence of a well defined upper rim of the acetabulum, are points that must influence the surgeon in his choice of arthrodesis or an attempt to retain a movable joint. Certainly with the pathology present there is more risk of recurrence of the dislocation if an attempt is made to preserve motion. In the majority of cases I have performed arthrodesis feeling that a stiff hip in good functional position would give great improvement and would obviate the risk of a recurrence of the dislocation incident to an attempt to retain motion.

Having decided on arthrodesis, the cartilage is removed from the head of the femur and from the acetabulum and the upper surfaces of these two structures are squared off so as to obtain a wide apposition of cancellous bone surfaces. Reduction is now secured by a slight pull on the leg and by pressure directly over the great trochanter. The ease with which the head of the femur is brought to the level of the acetabulum is a convincing demonstration of the effectiveness of skeletal traction in overcoming the shortening of the soft parts.

The area of contact between the head of the femur and the acetabulum can be increased by placing a bone graft in the angle between that part of the head projecting beyond the acetabulum and the denuded surface of the ilium lying directly above. Grafts may be secured from the crest of the ilium, but I usually take them from the great trochanter, fixing them in place by beef bone screws. The purpose of the graft is to increase the chances of obtaining bony ankylosis. Catgut sutures are placed in the capsule, muscle and fascia, while the skin is closed with silk. A plaster-of-Paris spica is applied with the hip held in a position of about 15 degrees of flexion, 15 degrees abduction and slight external rotation.

#### 4. POSTOPERATIVE CARE

The stitches are removed on the tenth day through a window in the cast. Fixation of the limb in a long spica is continued for at least four months. The time to begin exercises of the knee is determined by clinical and roentgen-ray examinations. It is important that the beginning ankylosis of the hip should not be disturbed through motion of the knee; these exercises must therefore be given by a skilled physiotherapist. When ankylosis has begun consolidation of the joint may be hastened by the use of a well fitted Thomas walking caliper splint. It has been my experience, however, that weight bearing is not desirable until six months after operation. I believe that in two of the cases cited below failure to secure bony ankylosis was caused by too early weight bearing. The caliper splint is worn for several months and then gradually discarded.

## RESULTS

Six patients have been treated by this method. The oldest was 16 years of age, the youngest, 10. Four of the dislocations were congenital in type, while the other two were due to residual paralysis of the gluteal muscles which followed an attack of anterior poliomyelitis. The longest period of observation following operation was three years; the shortest was eleven months. Arthrodesis of the hip was done in four cases and bony ankylosis was obtained in two; the other two retained a few degrees of motion. In two children the attempts to preserve motion were suc-

cessful, but one of these must be counted as a failure because, through error, the head of the femur was replaced in a false acetabulum. Of the five cases, therefore, in which reduction was obtained, two of the patients had bony ankylosis and three retained a small range of motion. All five showed stability of the hip and increased length of the leg. The results were not highly satisfactory in the movable hips, however, because of persistent limp and the development of adduction deformity. It seems likely that a failure to show a more complete correction of the lurching gait was due to coxa vara and a great weakness of the gluteal muscles. Correction of the coxa vara by osteotomy and exercises to strengthen the gluteal muscles might possibly lead to better results when motion is the aim of treatment.

The best functional results were secured in the cases in which bony ankylosis occurred. These hips are stable and in good position for weight bearing. The gait is strikingly improved and there is no tendency for adduction deformity to develop. From this series, therefore, it is quite logical to conclude that the most satisfactory functional results are obtained in the cases in which bony ankylosis is secured in a good position for weight bearing.

At the present time I have two other children in whom reduction was secured and motion preserved. An attempt was made to prevent recurrence by reconstructing a new shelving brim to the acetabulum through the use of bone grafts. Though of too recent date to permit of reporting end-results, these patients do not show the functional improvement seen in those children in whom bony ankylosis was secured.

## CONCLUSIONS

1. In old congenital dislocations of the hip it is difficult to secure reduction by the usual methods of treatment because of the marked pathologic changes that have taken place in the joint.

2. These pathologic changes consist of a structural shortening of the soft parts of the hip and thigh which fixes the head of the femur well above the acetabulum, and of a poorly developed acetabulum filled with fat and folds of capsule.

3. The condition in adult life is productive of severe disability and palliative measures give the patient but little relief.

4. Skeletal traction is effective in stretching the contracture of the soft parts and bringing the head of the femur to the level of the acetabulum. Reduction can then be secured by operation.

5. The decision at operation as to whether arthrodesis should be done or an attempt made to preserve motion must be made by a knowledge of results obtained by both methods.

6. In this series of five successful reductions the functional result judged from the standpoint of stability, increased length of the leg and improvement in gait was much the best in the cases in which a bony ankylosis in good position for weight bearing was obtained.

## REPORT OF CASES

CASE 1.—*History.*—A. M., a school girl, aged 11, was admitted to the University Hospital, Dec. 1, 1921, because of a marked limp in the left leg and shortening. The condition was first noticed when the child began to walk.

*Physical Examination.*—In walking there was a decided limp on the left side and measurements showed 2½ inches (6.2 cm.) shortening of the left lower extremity. The great trochanter was above Nélaton's line and motion of the left hip joint was limited in abduction, but free in all other directions. Trendelenburg's sign was positive. The Wassermann reaction on the blood was negative. Roentgen-ray examination, December 6, showed the head of the left femur displaced upward and backward and articulating in a false acetabulum (fig. 3). The diagnosis was congenital dislocation of the left hip.

*First Operation.*—December 8, under gas and oxygen anesthesia a linear incision was made just behind the anterior superior spine of the ilium. A subperiosteal reflection of the origin of the sartorius, tensor fascia femoris and rectus muscles was done. Through a stab incision a Steinman pin was inserted through the lower femoral shaft. (This operation was done to relieve the contracture of the flexor muscles.)

Twenty-five pounds traction was applied and maintained until Jan. 29, 1922, when roentgen-ray examination showed the head of the femur riding directly opposite the acetabulum. The Steinman pin was then removed.

*Second Operation.*—February 2, Kocher's posterior approach to the hip was used. The much thickened capsule was opened and the head of the femur was atrophic and joined to the shaft of the femur by a short, stubby neck. Coxa vara deformity was well developed. Arthrodesis was decided on and the cartilage was removed from the acetabulum and head of the femur. The dislocation was easily reduced and a small graft of cancellous bone taken from the trochanter was wedged between the upper surface of the head and the superior border of the acetabulum. The wound was closed in layers and a plaster-of-Paris spica was applied with the leg held in 20 degrees of flexion and 20 degrees of abduction.

The convalescence was uneventful and the wound healed by first intention. The patient wore the spica until June 1, 1922, when a walking caliper splint was applied and she began to bear weight. She wore this splint until October 5. Roentgen-ray examination, Sept. 17, 1923, showed a solid bony ankylosis (fig. 4).

Final examination was made April 3, 1924, and showed the hip to be stable with less than 1 inch (2.5 cm.) shortening of the limb. The patient walks well and with only a slight limp.

CASE 2.—*History.*—A. C., a girl, aged 10 years, was admitted to the University Hospital, Dec. 28, 1922, because of a limp and shortening of the left leg, which had started when the patient began to walk.

*Physical Examination.*—The patient walked with a distinct lurching gait and there was a well marked lumbar lordosis. There was 2½ inches (6.2 cm.) shortening of the left leg and the great trochanter was well above Nelaton's line. The motion of the hip joint was limited in abduction. Trendelenburg's

sign was positive and the Wassermann reaction on the blood was negative. Roentgen-ray examination, December 29, showed a posterior superior dislocation of the head of the left femur. The diagnosis was congenital dislocation of the hip (fig. 5).

*First Operation.*—Jan. 4, 1923, a Steinman pin was inserted through the lower end of the shaft of the left femur. Traction was applied and the weight was

Fig. 3 (case 1).—Congenital dislocation of the hip in a girl, aged 11 years, before Steinman pin traction and operation.

then gradually increased up to 30 pounds. Roentgen-ray examination, January 26, showed the head of the femur directly opposite the acetabulum (fig. 6). The Steinman pin was removed.

*Second Operation.*—January 30, the Smith-Peterson incision was used, and after a much thickened capsule was incised, the head of the femur and the upper border of the acetabulum were exposed. There was a coxa vara

deformity of the neck; the head of the femur was small and atrophic, and there was almost complete absence of the upper brim of the acetabulum. The cartilage was removed from the head of the femur and the acetabulum while the upper surfaces of these two structures were squared off so as to afford a wide apposition of cancellous bone surfaces. The dislocation was then reduced and a trochanteric graft was placed at the junction of the upper surface of the

Fig. 4 (case 1).—One and one-half years after reduction by operation; the joint is solidly ankylosed.

head with the upper rim of the acetabulum. The wound was closed in layers and a plaster-of-Paris spica applied.

The convalescence was entirely uneventful and the wound healed by first intention. The plaster spica was removed, April 11, and the patient was allowed to be up wearing a walking caliper splint. There were a few degrees of motion in the hip, and as this motion was painless, it was thought advisable to try to preserve it through exercise.

Fig. 5 (case 2).—Congenital dislocation of the hip in a girl, aged 10, before treatment.

Final examination, August 20, showed the patient walking with a decided limp and with a positive Trendelenburg sign. The patient had a few degrees of motion in all directions which was painless. There was 1¼ inches (3.1 cm.) shortening of the limb. Roentgen-ray examination, September 20 (fig. 7), showed reduction but failure to secure bony ankylosis. Improvement can be noted in stability and increased lengthening of the leg, but the functional result is disappointing, as the patient has a lurching gait.

Fig. 6 (case 2).—Appearance after Steinman pin traction on Thomas splint; the head of the femur is now directly opposite the acetabulum.

CASE 3.—*History.*—A. H., a girl, aged 13 years, was admitted to the University Hospital, Oct. 29, 1922, because of a limp in the left leg which dated from the time when she first began to walk.

*Physical Examination.*—The left lower extremity was 2 inches (5 cm.) short and the trochanter was above Nélaton's line. Trendelenburg's sign was positive.

The motion in the hip joint was free in all directions except abduction, in which it was markedly limited. Roentgen-ray examination, October 29, showed a posterior dislocation of the hip with considerable torsion of the neck of the femur on the shaft (fig. 8). The diagnosis was congenital dislocation of the left hip.

Fig. 7 (case 2).—Appearance after reduction; arthrodesis was attempted but failed.

*First Operation.*—October 31, a Steinman pin was inserted through the lower end of the shaft of the left femur. Traction of 15 pounds was applied and increased to 30 at the end of two weeks. December 5, measurements showed the legs to be the same length and roentgen-ray examination showed the head of the femur on a level with the acetabulum (fig. 9). December 9, the Steinman pin was removed.

*Second Operation.*—December 11, through a Smith-Peterson incision the capsule of the hip joint was opened showing the head and neck of the femur and the upper border of the acetabulum. The neck was much shortened and thickened and the acetabulum was shallow. The head of the femur was small and atrophic. The cartilage was then removed from the acetabulum and the

Fig. 8 (case 3).—Congenital dislocation of hip in girl, aged 13 years, before treatment.

head of the femur, and with an osteotome a small section of ilium was turned downward over the head of the femur. Against the raw surface of the ilium exposed by turning down this pedicle, a graft taken from the great trochanter was fixed in place by beef bone screws. The wound was closed in the usual manner and a plaster-of-Paris spica was applied with the limb held in about 25 degrees flexion and 25 degrees abduction.

The convalescence was uneventful and the wound healed by first intention. The inner Steinman pin wound required longer to heal than is ordinarily the case. Three months had elapsed before it was completely and firmly healed. The plaster-of-Paris spica was removed, March 12, 1923, about three months after operation, and there were a few degrees of motion present. Massage and exercises were begun in hopes that this motion could be increased. Roentgen-

Fig. 9 (case 3).—Appearance after Steinman pin traction on Thomas splint the head of the femur is directly opposite the acetabulum.

ray examination, March 12, showed reduction of the head, but a failure to obtain arthrodesis (fig. 10). Weight bearing with a Thomas walking caliper splint was begun April 15, and this splint was worn for a period of two months.

Final examination, August 20, showed the patient standing with a slight tilt of the pelvis and measurements showed 1 inch (2.5 cm.) shortening of the left leg. There was a considerable range of motion in the hip in all directions.

The hip joint was stable, but in walking a lurching gait remained. A roentgenogram taken August 20 showed reduction of the head.

This was the second case in which motion remained, and though the stability of the hip was improved, a lurching gait remained; the functional result was therefore not considered satisfactory.

Fig. 10 (case 3).—Appearance after reduction and reconstruction of the upper rim of the acetabulum by operation; the trochanteric graft is held in place by two beef bone screws.

CASE 4.—*History.*—V. D., a girl, aged 14 years, was admitted to the University Hospital, April 11, 1922, because of dislocation of the left hip. At the age of 3 years the patient had an attack of anterior poliomyelitis which left her with a paralysis involving the left hip and foot. She had had no treatment before admission to this hospital, except massage. She now complained of weakness, limp and shortening of the left lower extremity.

*Physical Examination.*—The left lower extremity showed a foot drop due to paralysis of the dorsiflexor muscles of the foot. There was a paralysis of the gluteus medius and the gluteus maximus muscles and the head of the femur was dislocated posteriorly and could be readily palpated in the buttock. There was 2¾ inches (6.8 cm.) shortening. Roentgen-ray examination, April 12, showed a posterior dislocation of the head of the femur (fig. 11). The diagnosis was dislocation of the left hip due to paralysis of the gluteal muscles.

Fig. 11 (case 4).—Paralytic dislocation of the hip in a girl, aged 14 years.

*First Operation.*—April 17, a Steinman pin was inserted through the lower end of the femur. Twenty-five pounds traction was applied and maintained until May 8, when roentgen-ray examination showed the head opposite the acetabulum.

*Second Operation.*—May 10, through a Smith-Peterson incision the capsule was incised and the head and neck of the femur were exposed. There was a

marked coxa valga deformity present. Cartilage was removed from the head of the femur and acetabulum and the dislocation reduced. A graft taken from the trochanter was transplanted against the denuded ilium in the manner described above. The wound was closed with interrupted sutures and a plaster-of-Paris spica applied with the limb held in 20 degrees of flexion and 20 degrees of abduction.

Fig. 12 (case 4).—Appearance five months after reduction and arthrodesis of hip; there is bony ankylosis.

The convalescence was uneventful and the wound healed by first intention. Fixation was continued in the plaster-of-Paris spica for six months and then weight bearing was allowed. Roentgen-ray examination, October 16, showed a firm bony ankylosis (fig. 12).

The patient was seen again, May 15, 1924, and a tendon fixation was done to correct the foot drop. At this time the hip was held in a position of about

20 degrees of flexion, 20 degrees of abduction and slight external rotation. There was a solid bony ankylosis. The patient walks well and sits with comfort and is entirely pleased with the result of treatment.

CASE 5.—*History.*—A. A., a boy, aged 16 years, was admitted to the University Hospital, June 13, 1921, because of a deformity of the left hip and a marked limp which had existed, according to his statements, from the time he began to walk.

*Physical Examination.*—The left lower extremity showed a marked shortening of the left leg with the trochanter elevated above Nélaton's line. There was quite pronounced atrophy of the thigh and of the calf. The foot was held in an equinovarus position with 40 degrees of flexion deformity of the hip. There was paralysis of the peroneal muscles and a weakness of the hamstrings and the gluteal muscles. The diagnosis was anterior poliomyelitis and posterior dislocation of the hip.

*First Operation.*—July 5, a straight incision was made just posterior to the anterior superior spine of the ilium. The origin of the tensor fascia femoris and the acetabular head of the origin of the rectus muscle were freed subperiosteally and the hip joint contracture was corrected by stretching. The wound was closed with catgut and silk. Calipers were inserted in the lower end of the femur in the usual manner.

After five weeks' traction roentgen-ray examination showed the head of the femur opposite the acetabulum and open reduction was decided on.

*Second Operation.*—August 11, through a Kocher's posterior approach the capsule of the hip joint was opened with exposure of the head of the femur and the acetabulum. The head of the femur was atrophic and irregular in outline, while the acetabulum was broad and shallow. The cartilage was removed from the acetabulum, but it was left intact on the head of the femur. Reduction was then effected, but the marked anteversion of the neck of the femur prevented replacement until the femur was rotated internally 90 degrees. The capsule was plicated with mattress sutures and the skin closed with silkworm-gut. A plaster-of-Paris spica was applied with the limb held in moderate abduction and extreme internal rotation, the patella facing directly inward.

The convalescence was uneventful and the wound healed by first intention. The patient was discharged from the hospital after two months. He returned again in January for correction of the internal rotation of the femur.

*Third Operation.*—Jan. 23, 1922, a linear incision was made on the lateral aspect of the lower third of the thigh. The muscles and the periosteum were divided and a transverse osteotomy was done. The lower fragment was then rotated outward 90 degrees. A plaster-of-Paris spica was applied.

The convalescence from the operation was uneventful and the wound healed by first intention. The patient was discharged wearing a walking caliper splint after three months in the hospital.

He was readmitted, September 21, for operation on the deformed foot when the insertion of the anterior tibial muscle was transferred to the outer side of the tarsus.

Final examination was made, July 14, 1924, three years after the reduction had been effected. The patient walked with the hip held in about 15 degrees of adduction and 30 degrees of flexion. Measurements cannot be accurately made on account of the deformity, but there is about 1½ inches (3.7 cm.) true shortening. The joint was not solidly ankylosed. This patient is an example

of reduction without securing a solid ankylosis. A definite deformity has developed and the limp is more marked than in those cases in which complete fixation of the joint was obtained.

CASE 6.—*History.*—T. J., a girl, aged 14 years, was admitted to the University Hospital, May 25, 1922, because of a limp in the left leg which had been present since she first began to walk. There was no complaint of pain in the joint, but there was pain in the low back after the patient had walked for long distances.

*Physical Examination.*—The patient walked with a decided limp in the left hip and the great trochanter was prominent. There was 2¼ inches (5.6 cm.) shortening. The head of the femur could be palpated in the buttock when the thigh was flexed and adducted. Roentgen-ray examination showed a dislocation of the head of the femur with considerable anteversion of the neck of the femur. The diagnosis was congenital dislocation of the left hip.

*First Operation.*—June 13, a Steinman pin was inserted through the lower end of the shaft of the femur. Twenty-five pounds of traction was applied and maintained until July 10, when roentgen-ray examination showed the head of the femur to be opposite the level of the acetabulum.

*Second Operation.*—July 12, through a Smith-Peterson incision the head of the femur and the acetabulum were exposed. The acetabular cavity was found to be filled with fibrous tissue and there was considerable difficulty in removing it. Reduction was effected and while the limb was held in abduction the wound was closed with catgut to the deep structures and silkworm-gut to the skin. A plaster-of-Paris spica was applied with the hip in about 30 degrees of abduction and 20 degrees of flexion.

Roentgenograms taken after operation showed that the head of the femur had been placed in a false acetabulum. The exercises were begun at the end of six weeks and the patient was allowed to walk on a walking caliper splint after a period of three months.

Final examination, Jan. 28, 1924, showed an excellent range of motion, but a positive Trendelenburg sign and a definite limp. There was 2 inches (5 cm.) shortening. The only improvement noted in this patient was some increase in stability of the joint.

NOTE.—The cases cited above were treated in 1921 and 1922. Since then a similar method of treatment has been described by N. Allison in an article entitled "Congenital Dislocation of the Hip in Young Adults" (J. Bone & Joint Surg. **7**:330 [April] 1925). In 1923 an article appeared by Dickson entitled "The Operative Treatment of Old Congenital Dislocation of the Hip." By his method traction was used preliminary to operation, but no attempt was made to reduce the dislocation.

# THE INFLUENCE OF DECOMPRESSION OPERATIONS ON EXPERIMENTALLY PRODUCED PAPILLEDEMA *

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Innumerable opportunities have been afforded clinicians to study the partial or total subsidence of papilledema produced by intracranial new growths after a subtemporal decompression had been performed. These observations have clinched the argument in favor of the mechanical theory of the production of choked disks. This operative procedure, first described by Cushing,[1] has had a definite influence on the surgery of intracranial tumors. The pendulum has swung from the improper and injudicious performance of a subtemporal decompression in every case of increased intracranial tension to the dogmatic statement that it should never be performed. The good results, as far as the subsidence of papilledema is concerned, are championed by all neurologic surgeons. On the other hand, Sachs[2] has stated that he has rarely seen any demonstrable benefit to the patient following a subtemporal decompression. Certain it is that this comparatively simple procedure has been done less frequently as our perfection in neurologic diagnosis has increased.

Curiously enough throughout the discussion of the value of decompression operations, and although no direct evidence is brought to bear, the suggestion runs that the decompression opening must be a local one. By this is meant that a subtemporal decompression is of no avail in the presence of an infratentorial new growth. It is assumed that the tentorium cerebelli predominantly, and the falx cerebri less so, resist the equal distribution of increased intracranial tension produced by the gradual enlargement of a new growth. Yet it is common experience that an intracerebellar tumor as a rule produces an early, rapid, and high degree of papilledema and so the increase in tension within the posterior fossa must be rather quickly transmitted to the structures in the anterior and middle fossae, including the optic nerves.

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1. Cushing, Harvey: Surg., Gynec. & Obst. **1**:287, 1905.

2. Sachs, B.: Shall We Decompress for Choked Disk? Arch. Neurol. & Psychiat. **8**:515 (Nov.) 1922.

All neurologic surgeons are in entire agreement that in the presence of any symptoms pointing to a definite localization, the surgical procedure should always be directed toward exposure and removal of the tumor. The infrequency of the performance of subtemporal decompressions will testify to the present high degree of diagnostic accuracy and to the custom of attacking the tumor. On the other hand it must be recognized that there are a certain number of cases in which all diagnostic procedures, including ventriculography, fail and in which the patient's vision must be saved while awaiting localizing symptoms.

It is felt that the subtemporal decompression has a definite, though limited, field of usefulness. It is questionable, however, whether the results of this operation are as pronounced in the presence of an infratentorial tumor as they are in the presence of a supratentorial new growth. Therefore, I have attempted to produce intracranial lesions that simulate new growths as closely as possible in the production of papilledema. Further, I have observed the effects of decompression operations on this experimentally produced papilledema.

## LITERATURE

Papilledema has been experimentally produced in many ways and the results of all these studies have emphasized the importance of increased intracranial pressure as the largest factor in its production. The mechanical, as opposed to the toxic, theory of the production of papilledema was first suggested by von Graefe [3] in 1860. After Schwalbe [4] had shown that the intravaginal lymph spaces of the optic nerve communicate with the cerebral subarachnoid spaces, Schmidt-Rimpler [5] and Manz [6] argued that an increase in intracranial pressure drove cerebrospinal fluid through the lamina cribrosa and produced an edema of the optic nerve sheath, which in turn compressed the vessels of the nerve and consequently resulted in an edema of the nerve head.

In 1909, Cushing and Bordley [7] presented the results of their experimental studies on the production of papilledema. They were able to produce an acute edema of the nerve head by injecting fluid into the subarachnoid space or by exerting digital pressure against the dura mater. They also injected melted paraffin into the cerebrum and obtained certain temporary retinal changes. However, the paraffin was evidently tolerated without any continued further effect other than the

3. Von Graefe: Arch. f. Ophth. **7**:56, 1860.
4. Schwalbe: Arch. f. mikr. Anat. **6**:1, 1869.
5. Schmidt-Rimpler: Arch. f. Ophth. **15**:193, 1869.
6. Manz: Deutsch. Arch. f. klin. Med. **9**:339, 1872.
7. Bordley, James, Jr.; and Cushing, Harvey: Observations on Choked Disk, J. A. M. A. **52**:353 (Jan. 30) 1909; Bull. Johns Hopkins Hosp. **20**:95, 1909.

destruction of cerebral tissue. Realizing that their experimental method did not simulate the mechanics of the production of papilledema by an intracranial new growth closely enough, they introduced fragments of sponge tents between the dura mater and the skull. By this means they were only partially able to realize their object, but in all events

Fig. 1 (Series 1, Dog 4).—Intracerebral tumor with partial occlusion of lateral ventricle.

they were quite able consistently to produce the appearance of papilledema by a mechanical increase in intracranial pressure. They did not record observations on the effect of decompression operations on the papilledema thus produced, although of course they had made many clinical observations of its efficacy.

In 1916, Parker [8] by similar experimental methods corroborated these findings both in monkeys and in dogs. He also established the fact that elevation of the nerve head occurred first in the eye that possessed the least intra-ocular tension. Kornder [9] produced hydrocephalus in dogs by blocking the cerebral aqueduct with paraffin. His animals showed papilledema that developed less acutely than that in the animals of the previous workers did and he believed that this method of experimentation most closely simulated the intracranial mechanics of a new growth. While many intracranial tumors are accompanied by a unilateral or bilateral dilatation of the lateral ventricles, it would seem that there may be other means of more closely simulating the actual pathologic condition and mechanics involved.

## METHODS

Thirty-five animals (dogs) were operated on in this experimental study. In the first and second series of animals intracerebral and intracerebellar tumors [10] were created. A small opening was made through the bone with a dental burr and a slit made in the dura mater through which the foreign body was introduced. The openings in the dura mater or in the bone were wholly insufficient in themselves to allow of any decompressive effect. In the third and fourth series of animals a classical subtemporal decompression operation was performed and at a later date an intracerebral tumor was created in the opposite hemisphere; or, the foreign body was introduced into a cerebellar hemisphere under the same circumstances. In the last group of animals, an intracerebellar tumor was created after a typical suboccipital decompression had been performed.

Many substances were used in an endeavor to find an inert body which would be capable of a subsequent increase in size and which would possess some elasticity to simulate the mechanical action of a new growth. It was found that gelatin would take up water, increase in size and then dissolve. Melted paraffin proved to be a substance which could not be well controlled and which proved to be as disadvantageous as Cushing and Bordley had said. Laminaria or sea sponge tents were used and proved fairly satisfactory. They were, however, difficult to sterilize.

Finally, agar-agar, sterilized by dry heat, was placed in gelatin capsules that had been sterilized in absolute alcohol and rinsed in sterile water. These capsules of agar were introduced into the brain substance

8. Parker, W. R.: Choked Disk, J. A. M. A. **67**:1053 (Oct. 7) 1916.

9. Kornder, L. H.: Hydrocephalus and Choked Disk, Arch. Int. Med. **23**:197 (Feb.) 1919.

10. The word "tumor" is used here simply to convey the idea of the swelling of a substance not normal to the part.

wherever desired. It was found that after a number of preliminary operations had been performed a 2 grain (0.13 gm.) capsule of agar would have taken up its largest amount of water at the end of from forty-eight to seventy-two hours, but even after that there was a very slow increase in the size of the mass. The gelatin capsule at body temperature dissolved rapidly leaving the agar quite unaffected. Agar of course is quite chemically inert. A larger quantity of agar, with the consequent production of a larger tumor mass, was never well borne by the dogs and an acute edema of the papilla with rapid death always

Fig. 2 (Series 1, Dog 4).—Section of optic nerve showing an absence of the physiologic cup and marked interfascicular edema.

resulted. The 2 grain capsule of agar produced a papilledema which would become evident after thirty-six hours and which would gradually increase, accompanied by evident symptoms of increased intracranial tension.

The subtemporal decompression operations were performed from two to three days before agar was introduced, rather than afterward, because many animals with an increased intracranial tension were lost as a result of the anesthetic.

Ophthalmoscopic examination of the eye grounds was made several times before the experiments were begun to become acquainted with the normal picture of the particular dog's disks. They were continued at frequent intervals throughout. After the animal was killed, the eyes and brain were fixed in situ with 10 per cent. formaldehyd solution.

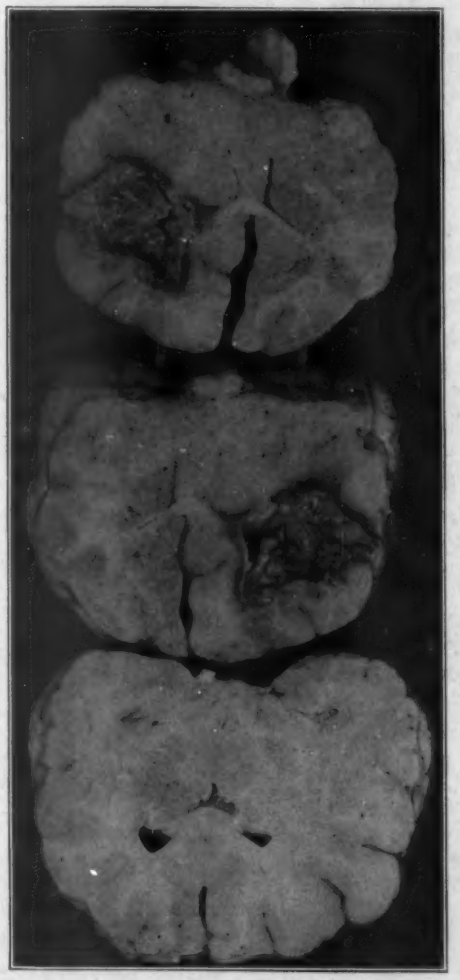

Fig. 3 (Series 1, Dog 2).—Intracerebral tumor which occupies a portion of the lateral ventricle.

The nerve heads and retinas were sectioned and stained with hematoxylin-eosin. The brains were sectioned coronally, mounted and the lesion photographed. In several animals the optic nerves at their entrance into the bony optic foramina were removed and after decalcification were sectioned. Sections of the tumor and the surrounding brain

substance were made and stained by Perdrau's method for connective tissue. Other studies are to be made of the microscopic changes in the vicinity of the tumor mass by the use of the silver impregnation methods.

The protocol of an animal typical of each of the five series of experiments will be given in addition to the general results obtained in each particular group.

Fig. 4 (Series 1, Dog 2).—Section of optic nerve which shows edema of nerve fibers and marked lateral displacement of the rods and cones.

### SERIES 1. INTRACEREBRAL TUMORS

The capsule of agar was usually placed in an area on the dorsolateral surface of the cerebral hemisphere corresponding to the posterior portion of the frontal lobe in man. The animals usually recovered quite promptly from the anesthetic. After from thirty-six to forty-eight hours the symptoms of increased intracranial tension became manifested. The animals would become stuporous, difficult to stimulate and would vomit. In two of the seven animals of this group, infection occurred and death resulted from a meningitis that was accompanied by convulsions, opisthotonos and rigidity. In three of the remaining animals a

contralateral hemiparesis developed. In each instance a definite elevation of the optic nerve head of from 3 to 4 diopters was measured.

The microscopic picture of the nerve head and retina showed an interfascicular edema and obliteration of the optic cup. In almost every case the layer of rods and cones could be seen to be pushed laterally by the swollen nerve head.

The size, shape and location of the tumors produced can best be realized by Figures 1, 2, 3 and 4. The microscopic appearance of a cross section of a normal dog's optic nerve head is seen in Figure 5.

Fig. 5.—Optic nerve and retina of normal dog; the larger of rods and cones at the margin of the optic nerve and compactness of the optic nerve fibers should be noted.

EXPERIMENT 1.—Dog 3, a brown female, was operated on at 2 p. m., Nov. 6, 1924, under ether anesthesia. A small burr opening about 5 mm. in diameter was made through the right parietal bone after this had been exposed through a vertical incision into the temporal muscle. The dura mater was slit longitudinally and a small wound made in the cerebral substance. A 2 grain gelatin capsule filled with agar was placed into the cerebrum at a depth of about 2 cm. The temporal muscle, fascia and scalp were closed carefully in layers.

At 4 p. m. the dog was moving about the cage but had not fully recovered from the anesthesia.

November 7, the animal had a definite left hemiparesis which produced a distinct ataxia. The dog had vomited twice without any retching or apparent effort. The retinal veins were engorged and the disk edges were not as distinct as previously.

November 8, the animal was very drowsy and was difficult to stimulate. She moved about slowly, awkwardly and with the head drooping. Water and food

Fig. 6 (Series 1, Dog 3).—Intracerebral tumor with unilateral dilatation of the opposite ventricle.

had been given through a stomach tube but the animal vomited three times afterward. It was typically projectile in character.

The morning of November 9 the animal died with a slowly decreasing pulse and respiratory rate.

At necropsy the carotid arteries were injected with 10 per cent. formaldehyd and the brain and orbits were removed. Figure 6 shows the cerebral lesion that

had been produced. The size of the mass can readily be seen, as well as its relation to the right lateral ventricle. The partial obliteration of the ventricle on the same side with a corresponding enlargement of the opposite ventricle is apparent.

Figure 2 shows a marked interfascicular edema. The subarachnoid space surrounding the nerve is distended. The optic cup is absent and the layer of rods and cones is pushed laterally.

SERIES 2. INTRACEREBELLAR TUMORS

The agar capsule was placed within the cerebellar hemispheres or within the vermis. In the former event a definite ataxia, dysmetria,

Fig. 7 (Series 1, Dog 3).—Section of optic nerve showing interfascicular edema; rods and cones are pushed laterally.

progression in a circle, and persistent turning of the occiput toward the side of the lesion were the most common symptoms. In five of the seven animals a temporary early nystagmus was produced but in each case this quickly disappeared. In two of the animals the tumor was located in the vermis. These animals showed no incoordination or ataxia other than the general weakness that accompanied the terminal increase in intracranial tension.

In all these animals a definite papilledema could be seen and measured. The clinical picture of an advanced choked disk with flamelike hemorrhages and an exudate about the vessels was not seen. The dogs gradually became stuporous, hard to arouse and died or were sacrificed after from seventy-two to ninety-six hours.

Fig. 8 (Series 2, Dog 18).—Intracerebellar tumor.

EXPERIMENT 2.—Dog 18, a large Airedale, was operated on at 1 p. m., Jan. 9, 1925, under ether anesthesia. A cross-bow incision was made in the suboccipital area and the suboccipital bone was uncovered. A small opening through the bone was made with the dental burr. The dura mater was slit open and a 2 grain capsule of agar was introduced into the right cerebellar hemisphere. The wound was closed in layers.

At 3 p. m. the animal was turning over and over in the cage in an effort to right itself.

January 10, the animal stood with difficulty with the legs far apart. The occiput was pulled downward and to the right. There was a slight nystagmus with the quick component to the right. The animal could not walk without falling down on the right side. The disks showed venous engorgement and an elevation of 2 diopters.

January 11, the animal had not taken food or drink and lay in a semiconscious state. He could be aroused only by strong stimuli and was quite unable to stand. There was a definite papilledema with blurred disk margins.

Fig. 9 (Series 2, Dog 18).—Section of nerve head which shows interfascicular edema and lateral displacement of the rods and cones.

January 12, the animal died during the afternon. The brain and orbits were removed after fixation. The cerebellum showed a large mass that occupied the dorsal portion of the right cerebellar hemisphere. The mass forced the lingula of the vermis down onto the floor of the fourth ventricle and produced a partial obliteration of that cavity. There was a moderate degree of dilatation of the lateral ventricles. The microscopic picture of the optic nerve heads was that of a marked edema that separated the nerve bundles. The rods and cones were separated lateralward and the nerve head was bulging. The subarachnoid space was distended (Figs. 8 and 9).

A section of the cerebellum taken through the mass and including a portion of the cerebellum was stained by Perdrau's method for connective tissue (Fig. 12). The large strands of connective tissue within the cerebellar cortex were typical of the changes that took place about this mass of agar. Hematoxylin-eosin stains showed a large proliferation of round cells about the mass. Further stains with the silver impregnation methods of Cajal and Hortega are in progress.

Fig. 10 (Series 2, Dog 20).—Intracerebellar tumor situated near the midline.

Figures 10 and 11 show another tumor mass with the resulting papilledema. This mass was placed in the midline within the vermis. This animal showed no ataxia and no nystagmus but presented the symptoms of increased intracranial tension.

SERIES 3. INTRACEREBRAL TUMORS WITH A SUBTEMPORAL DECOMPRESSION

After a subtemporal decompression had been performed and the animals had recovered completely, a 2 grain capsule of agar was placed into the opposite cerebral cortex. Seven animals so operated on form Series 3. A papilledema developed in two of these animals, in which a 5 grain (0.32 gm.) capsule of agar was introduced. It was apparent that the size of the decompression opening was insufficient to take care of the swelling and increase of tension produced by this large mass

Fig. 11 (Series 2, Dog 20).—Section of optic nerve head showing interfascicular edema and lateral displacement of the rods and cones.

of agar. A larger decompression opening in the remaining five animals was in every case sufficient to prevent the occurrence of papilledema from the introduction of a 2 grain capsule of agar. It will be remembered that this amount of agar invariably produced papilledema in the absence of a decompression opening. Of course this fact has been observed clinically in the presence of a large infiltrating tumor, which produces a high degree of edema with an improperly placed or small decompression opening.

These five animals all recovered from the implantation of the agar and all showed varying degrees of motor involvement on the opposite side. All these animals lived from one to four weeks, however; at the end of this interval they were killed. Such animals could be returned to the general animal cage without difficulty and were quite able to care for themselves.

EXPERIMENT 3.—Dog 15, a large black male, was operated on, Dec. 20, 1924, under ether anesthesia. A left subtemporal decompression was performed in the usual manner and a cranial opening about 3 cm. square was made. The temporal

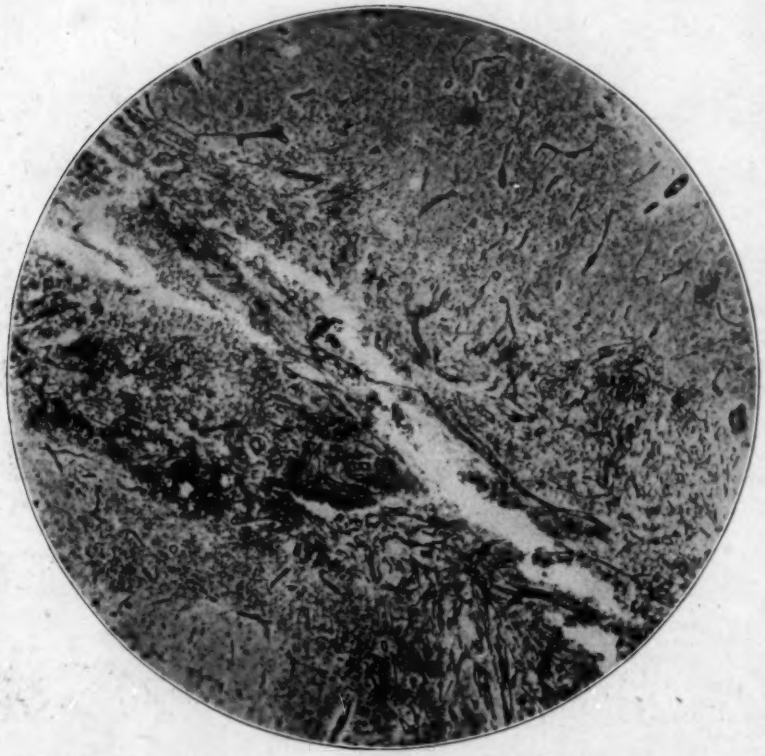

Fig. 12.—Section of cerebellum surrounding tumor mass in Dog 22; abundance of connective tissue fibers; Perdrau's stain.

muscle and fascia were carefully closed in layers after the dura mater had been opened with a stellate incision. The animal recovered quickly from the anesthetic and showed no evidences of cerebral injury.

December 23, the animal was bright and alert. A 2 grain capsule of agar was introduced into the right cerebral cortex in the usual manner. The animal recovered from the anesthetic in a short time.

December 24, the animal walked about but showed a weakness of the left hind and forelegs. He took food and water without difficulty. The optic disks were quite unchanged.

December 26, the animal walked about the room with a definite ataxia, which was due to his left sided weakness. The veins of the retina were normal and the disks were not elevated.

Jan. 2, 1925, the animal had been in the general dog room for a week. He was quite happy and active. The optic disks were normal and the left sided weakness had greatly improved.

January 4, the animal was killed and the brain and eyes were fixed in formaldehyd. The lesion found in the cerebral hemisphere was a narrow one extending to and compressing the lateral wall of the ventricle so that its cavity was obliterated. The consequent dilatation of the opposite ventricle on the side of the decompression may be seen in Figures 13 and 14. The microscopic sections of the optic nerve head and retina showed no swelling of the nerve and no edematous involvement of the fibers of the nerve. The layer of rods and cones were in their normal relation to the edges of the nerve as it enters the retina.

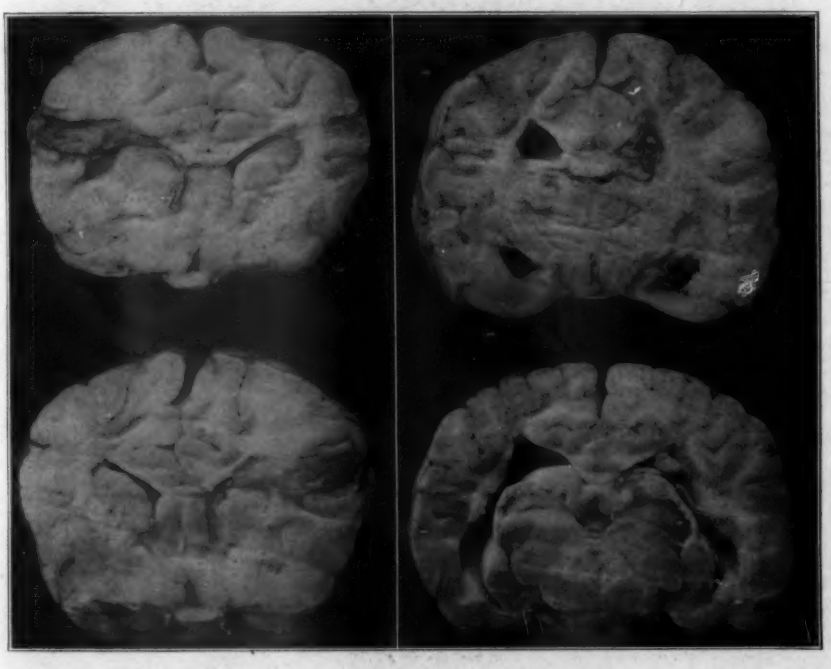

Fig. 13 (Series 3, Dog 15).—Intracerebral tumor with subtemporal decompression; unilateral dilatation of lateral ventricle.

Figures 15 and 16 show the coronal sections and section of the nerve head of Dog 17, another animal belonging to this group. Here again may be seen the presence of a unilateral hydrocephalus with the absence of a papilledema.

### SERIES 4. INTRACEREBELLAR TUMORS WITH A SUBTEMPORAL DECOMPRESSION

The same operative procedure was employed on the animals in Series 4 as was used in the preceding group. Three of the seven animals of this group died of an infection following the preliminary

subtemporal decompression operation. In the remaining four animals a 2 grain capsule of agar was introduced into the cerebellar hemispheres or vermis through an opening in the bone and dura quite insufficient to afford a decompression. In none of these four animals were we able to detect any papilledema by ophthalmoscopic examination. Three of the animals showed a marked ataxia and incoordination and in the fourth the tumor was confined to the vermis. These animals recovered well from their secondary operations. It required all of them from

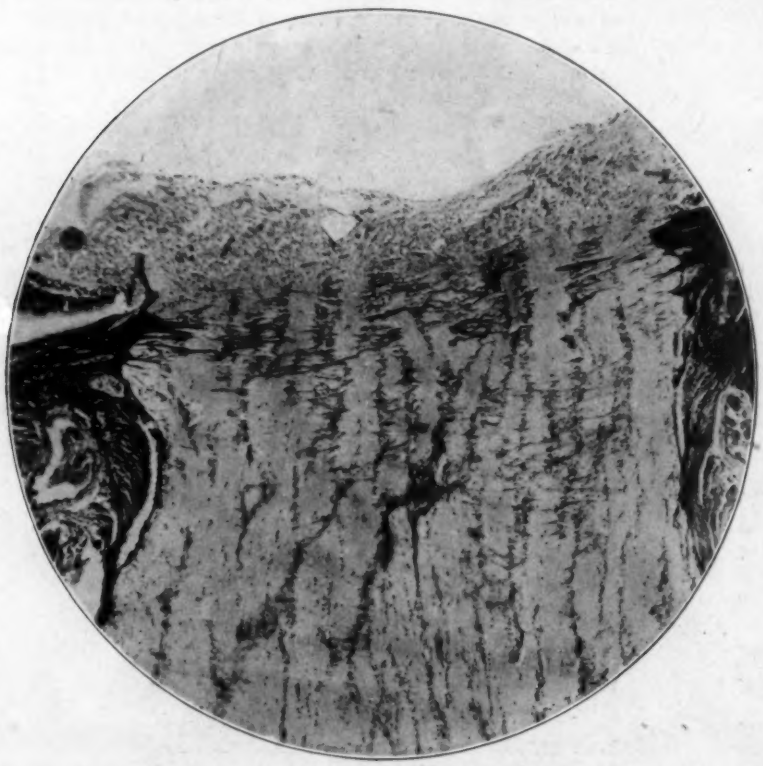

Fig. 14 (Series 3, Dog 15).—Intracerebral tumor showing absence of interfascicular edema; rods and cones in normal position.

three to four days to compensate partially for their cerebellar lesions so that they could move about. All these animals were killed at varying intervals of from one to two weeks.

EXPERIMENT 4.—A right subtemporal decompression was performed on Dog 23, a medium sized black animal, March 2, 1925. A bony opening about 3 cm. in diameter was followed by a star shaped dural incision. The wound was carefully closed.

March 6, the animal had well recovered from his previous operation and was eating well. He was quite alert and active. A 2 grain capsule of agar was

introduced into the midline of the cerebellar vermis. A very small opening was made in the bone and dura just large enough to admit the capsule. The animal lay normally and carried his head in the median line as he recovered from the anesthesia.

March 8, the animal walked about the room with the head held close to the ground and with a swaying, lolling gait. There was no progression to one side or the other. The retinal veins were in the same condition as before operation without any engorgement.

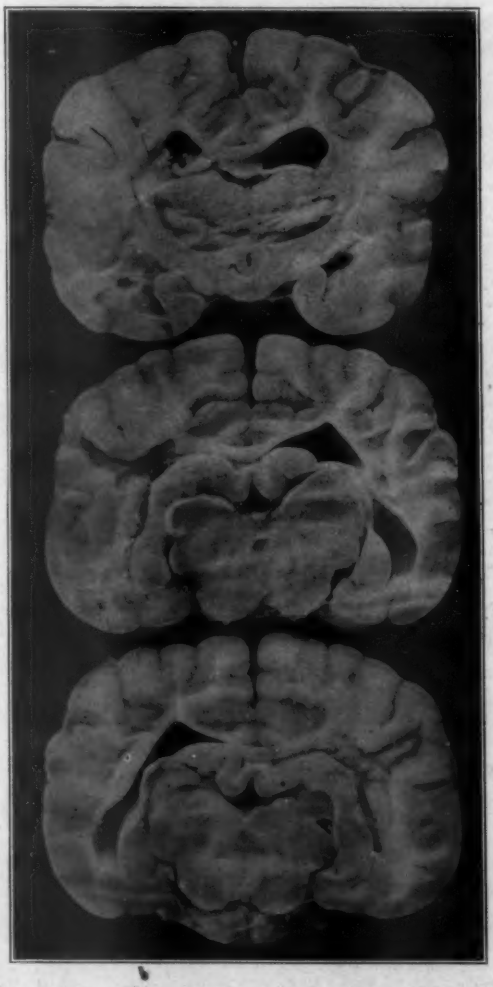

Fig. 15 (Series 3, Dog 17).—Intracerebral tumor with subtemporal decompression; unilateral dilatation of lateral ventricle on opposite side.

March 10, the retinas were still without changes. The disk edges were quite clear and the veins were not engorged or tortuous.

March 15, there had been no change in the ophthalmoscopic findings. The animal was sent back to the general dog cages and took care of himself very well. He was killed.

A definite, well marked and large tumor mass was found in the cerebellum slightly to the right of the midline of the vermis. There was a moderate dilatation of the lateral ventricles and the cerebral aqueduct and a bulging of the cerebral hemisphere through the decompression opening.

Figures 17 and 18 show coronal sections of the brain and a microscopic section of the optic nerve head of this animal. There was no microscopic evidence of papilledema. The subarachnoid space was normal in its size and relation to the nerve. The layer of rods and cones was in normal position.

Figures 19 and 20 show the brain and optic nerve head of Dog No. 22; here there is a more marked bulging of the cerebral cortex as well as a more pronounced dilatation of the lateral ventricles.

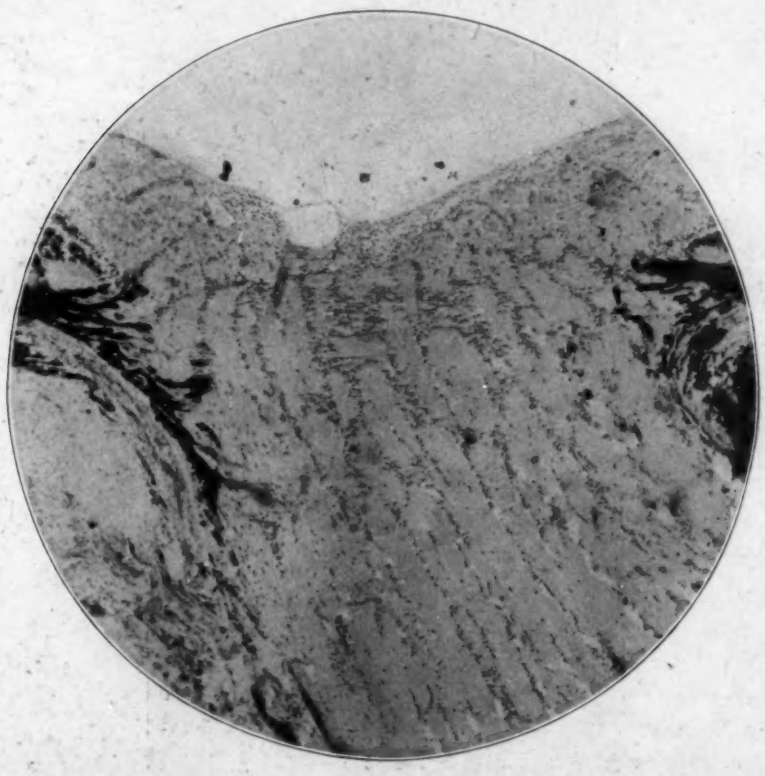

Fig. 16 (Series 3, Dog 17).—Intracerebral tumor showing absence of choked disk.

### SERIES 5. INTRACEREBELLAR TUMORS WITH A SUBOCCIPITAL DECOMPRESSION

In the seven animals in Series 5 a suboccipital craniotomy was performed and the cerebellar hemispheres and vermis were widely exposed. The dura mater was opened and the capsule of agar was introduced into the cerebellar hemisphere or vermis as the case might be. In these animals the suboccipital decompression operation and the implantation of the mass of agar were accomplished during the same operation. In

three of the animals of this series an infection occurred with a resulting meningitis. Observations on these animals were discarded. All of the four remaining animals recovered promptly from the operation. In two of these dogs the agar was introduced into the vermis and these animals recovered much more quickly than did the two in which the lesion was in the right or the left cerebellar hemispheres. It required two or three

Fig. 17 (Series 4, Dog 23).—Intracerebellar tumor with subtemporal decompression; dilatation of lateral ventricles.

weeks for the latter to compensate sufficiently from their ataxia and dysmetria to move about. None of these animals showed any evidence of increased intracranial tension. The ophthalmoscopic picture was quite normal at all times. As far as we could determine this group of animals acted exactly as did the group with cerebellar tumors in which a subtemporal decompression had been performed.

EXPERIMENT 5.—Dog 25, a large adult male, was operated on March 10, 1925, under ether anesthesia. With a cross-bow incision the large group of neck muscles were detached from their insertion to the suboccipital ridge. The suboccipital bone was entirely rongeured away and the dura mater over both cerebellar lobes and the vermis was uncovered. The dura was widely opened and a 2 grain capsule full of agar was introduced into the vermis of the cerebellum. The muscles were closed carefully in layers. The animal recovered very well from the anesthesia and was able to maintain an upright position.

March 12, the animal was moving about and ate and drank if he was allowed to lie down. The animal was alert and showed no evidence of increased intracranial tension. The vessels of the retina appeared unchanged, and there was no elevation of the disks.

Fig. 18 (Series 4, Dog 23).—Intracerebral tumor showing absence of interfascicular edema and normal position of rods and cones.

March 15, the animal walked about the laboratory wtih an ataxia and deviation to the right; he ate well and had shown no evidence of increased tension. The optic disks showed no elevation and the margins of the nerve head were clearly defined.

March 17, the animal was killed and the brain and orbits were removed. When the brain was sectioned a circumscribed mass was seen to occupy a portion of the vermis and lay slightly to the right of the median line. There was a moderate dilatation of the lateral ventricles. The microscopic sections of the nerve head and retina showed no evidence of a papilledema. The rods and cones

of the retina were in their normal position and showed no lateral displacement from the edges of the optic nerve. There was no evidence of an interfascicular edema (Figs. 21 and 22).

SUMMARY OF RESULTS

A definite tumor mass was created in these series of animals by the introduction of a 2 grain gelatin capsule of agar into the cerebral or cerebellar substance. Agar, by its tendency to take up fluid from the brain tissue, increases in size and gradually produces a rise in intracranial tension sufficient to create a papilledema and other symptoms

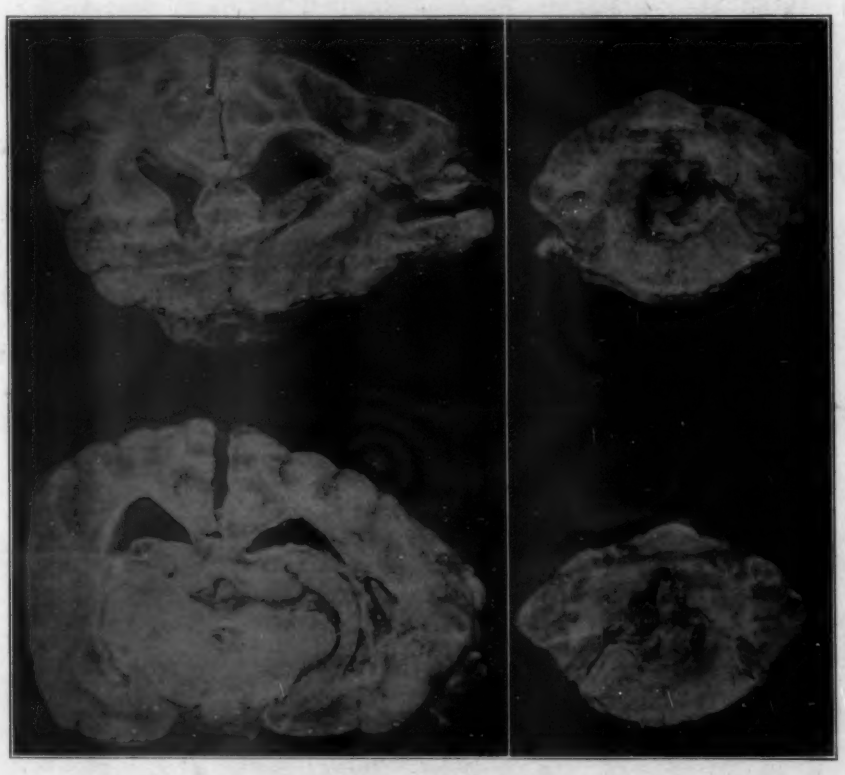

Fig. 19 (Series 4, Dog 22).—Intracerebellar tumor with subtemporal decompression; marked dilatation of lateral ventricles.

of increased intracranial tension, such as drowsiness, stupor and projectile vomiting. Papilledema did not develop in any of the animals on which a decompression operation had been performed. This was true when a subtemporal decompression was performed in the presence of either a supratentorial or an infratentorial lesion. There proved to be no difference between the effects produced by a subtemporal or a suboccipital decompression opening in the presence of a cerebellar tumor mass. The implantation of agar into the cerebral hemisphere was often

accompanied by a unilateral hydrocephalus in the lateral ventricle opposite to the side of the tumor. A bilateral hydrocephalus was often produced as a result of obstruction due to the implantation of a cerebellar tumor mass. In either event the effect of the subtemporal decompression opening was such as to prevent the occurrence of a papilledema or other symptoms of increased intracranial tension.

## COMMENT

The purpose of this work has not been to hold a brief for the performance of decompression operations in the surgical treatment of

Fig. 20 (Series 4, Dog 22).—Section of optic nerve showing absence of choked disk.

intracranial tumors. We strongly believe, as do all neurologic surgeons, that in the presence of symptoms of the slightest localizing value, an osteoplastic flap or a suboccipital craniotomy should be performed in an endeavor to attack directly the new growth. The infrequency of subtemporal decompressions done in any of the neurosurgical clinics testifies to the present day accuracy in the localization of intracranial tumors. On the other hand, it must be realized that there is a group

of patients in whom no localizing symptoms may be present and on whom all methods of diagnosis, including the mechanical aids, fail to give a surgical lead. Following a subtemporal decompression, every neurologic surgeon has had the experience of observing a subsidence of the choked disks in these patients; he has seen them relieved of terrific

Fig. 21 (Series 5, Dog 25).—Intracerebellar tumor with suboccipital decompression.

headaches and returned to a useful position in society while he has carefully watched for the appearance of the first symptom of localizing value.

Such clinical experiences have been said to be the rule only in tumors later verified as supratentorial in location. However, the same

results have been observed following a subtemporal decompression for the relief of symptoms of increased intracranial pressure in unlocalizable tumors that later have been verified as infratentorial in position. It must be emphasized that many decompression operations are performed in the presence of an advanced optic atrophy; they may be placed faultily or they may be performed when the lesion should have been directly attacked. Such facts must be taken into consideration in evaluating the results of the operation.

Fig. 22 (Series 5, Dog 25).—Section of optic nerve showing absence of interfascicular edema.

Certainly the results of these experiments on animals would lead one to the belief that a supratentorial decompression opening is just as effective in relieving or preventing papilledema in infratentorial as in supratentorial tumors. Whether the headache may be relieved to the same degree as papilledema may be questionable, since local tension on the dura mater by the immediately adjacent tumor may be a factor of considerable importance in the production of the headache. It is difficult to see why an internal hydrocephalus due to an infra-

tentorial lesion or the tentorium cerebelli should vitiate the decompressive effect of a cranial opening above the tentorium. Both these factors were present in several of the experimental animals without interfering with the mechanical effect of the subtemporal decompression opening.

The failure to realize the importance of the mechanical increase in intracranial pressure as a factor in the production of papilledema obstructed the acceptance of the subtemporal decompression operation as a sound surgical therapeutic principle in a limited group of cases. The realization that intracranial pressure is distributed equally throughout the supratentorial and the infratentorial fossae should do much to offset the view that a subtemporal decompression is of no value in the presence of an infratentorial tumor.

By the method of implantation of capsules of agar into the brain substance it is felt that a means has been found by which many of the effects of intracranial new growths may be simulated. The symptoms of disturbed function produced by the introduction of this material into various anatomic divisions of the cerebellum has been particularly instructive. The same is true of the microscopic investigation of the changes that occur in the brain tissue immediately surrounding these tumor masses.

## CONCLUSIONS

1. The introduction of sterile agar into the cerebral or the cerebellar substance produces an increase in intracranial pressure which results in the production of papilledema.

2. The gradual increase in size of the agar due to its absorption of fluid closely simulates the mechanics of an intracranial new growth.

3. A subtemporal decompression opening prevents the occurrence of papilledema from the introduction of agar into the cerebrum or the cerebellum. Such a cranial opening is as efficient in the presence of a cerebellar tumor as is a suboccipital craniotomy.

4. The importance of the mechanical factor of an increase in intracranial pressure in the production of papilledema has been corroborated by this method.

5. Connective tissue in abundance is found in the tissue immediately surrounding these artificially produced tumor masses.

6. This method of experimentation affords a means of studying the disturbed function of the cerebrum and the cerebellum produced by the introduction of agar into the brain substance.

## DISCUSSION

DR. JULIUS GRINKER, Chicago: This paper applies to the cases that have not yet developed a complete choked disk. I should like to ask Dr. Davis whether he was able to cause a subsidence of the choking of the disk. A number of

years ago I was interested in a case that I diagnosed as cerebellopontile angle tumor. But the surgeon to whom I referred the case was not convinced. He suggested a double decompression operation, to which I consented because of his unwillingness to operate while the patient was getting blind from the atrophy accompanying the choked disk and before there was complete secondary optic atrophy. The operation was done but the patient became blind.

Subsequently another surgeon removed part of the tumor from the cerebellopontile angle recess after which the patient lived four years. The postmortem examination revealed a large tumor. I believe that if the kind of decompression that Dr. Davis did experimentally could have been done in this boy's case, he would not have become blind. The optic atrophy proceeded as undisturbed as though there had not been a decompression. I take it that Dr. Davis has in mind only the cases that have not yet developed choked disk; in other words, that as a prophylactic of choked disk it may be a splendid procedure. Unless a patient can be assured, however, that he is going to get relief as far as vision is concerned, I do not think he will be willing to subject himself to a decompression operation.

Dr. T. B. Throckmorton, Des Moines, Iowa: In some of the slides that have been presented I noticed that when the tumor had been produced artificially and no decompression had been done, choked disk inevitably occurred. In other cases in which the tumor had been produced artificially and decompression was done, no choked disk occurred. I should like to ask Dr. Davis to explain when the decompression was done, whether it was done at the time of introduction of the capsule or whether it was done hours or days later.

Dr. Loyal Davis, Chicago: I have endeavored to show that an accurately placed and properly performed subtemporal decompression is of avail in increased intracranial tension regardless of the location of the tumor with reference to the tentorium cerebelli. Certainly, there are a number of cases on record in which a subtemporal decompression was done before the patient showed any localizing symptoms, and then when he did show localizing symptoms, an intracerebellar tumor was later removed. When we speak of subtemporal decompressions, we must realize that there are several kinds of operations performed in spite of the classical description first given by Cushing. Some are faultily placed; others are not sufficiently large to be a decompression and even some are so called when the dura is not opened. Therefore, when we speak of the results of subtemporal decompressions in the presence of intracranial tumors, we must be absolutely sure that there is an adequate decompression. With reference to these experiments, the decompressions were done both before and after the choked disks had appeared. I found that from seventy-two to ninety-six hours was the optimal time within which a decompression could be done on an animal, because with the increasing pressure the animal's physical condition begins to suffer and then when it is subjected to another operative procedure, with the administration of an anesthetic, it is likely to be fatal. I wish to emphasize again that subtemporal decompressions are now rarely done among neurologic surgeons since the percentage of accurate localizing diagnoses is much higher. On the other hand, in spite of all methods of diagnosis, including ventriculography, there are a few instances in which the patient is most benefited by a properly performed subtemporal decompression. In such instances I feel that such an operation is of value regardless of the relation of the tumor to the tentorium cerebelli.

# THREE TUMORS ARISING FROM NEUROBLASTS*

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The neuroblastoma, a tumor arising from neuroblasts or primitive nerve cells, although still somewhat of a pathologic rarity, is commoner than used to be supposed. Many cases described in the literature as sarcoma of the suprarenal or of the liver, congenital tumor of the suprarenal with metastases in the liver, lymphosarcoma of the suprarenal, malignant hypernephroma of the suprarenal medulla, etc., are now known to have been examples of neuroblastoma. Moreover, it is more than probable that the numerous cases reported as retroperitoneal sarcoma in children are also to be included in this category. Wright [1] saw five instances of the disease in a single year, and I have recently encountered four in a similar time. A necropsy was obtained on three of these, the results of which are presented here.

It was Virchow, in 1864, who first guessed at the nervous tissue origin of these congenital suprarenal tumors. He made the mistake of regarding them as gliomas, a mistake that has been repeated at frequent intervals ever since. In 1891 Marchand pointed out the resemblance between the structure of the tumor and that of the developing sympathetic system, and suggested that the tumor was derived from neuroblasts, thus for the first time placing our knowledge of the disease on a firm foundation. Since that date reports of a considerable number of cases have appeared, chiefly in the German literature, under a variety of names, such as neuroblastoma, neurocytoma, sympathicus tumor, ganglioneuroma, etc. To classify as neuroblastomas all tumors in children arising from the suprarenal medulla or from the sympathetic nervous system is, however, no longer sufficient. Neuroblasts may develop either into sympathetic ganglion cells or into the chromaffin cells of the paraganglions, such as the suprarenal medulla and the carotid body. It is thus evident that one of these tumors may originate in three ways: (1) from the original neuroblasts, a neuroblastoma; (2) from the ganglion cells, a ganglioneuroma, and (3) from the chromaffin cells of the paraganglions, a paraganglioma or chromaffinoma.

Of these the neuroblastoma proper is by far the most common. It is composed of small round cells with dark hyperchromatic nuclei, closely resembling the cells of a small round cell sarcoma. From the

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1. Wright, J. H.: Neurocytoma or Neuroblastoma, a Kind of Tumor Not Generally Recognized, J. Exper. Med. **12**:556, 1910.

latter it may be distinguished by the presence of fibrils that do not give the characteristic staining reaction of collagen, fibroglia or neuroglia. They are arranged in longitudinal bundles and occasionally in rounded balls or masses around which the neuroblasts are grouped so as to give the appearance commonly known as a rosette. The absence of rosettes, however, does not negative a diagnosis of neuroblastoma. The fibrils must be regarded as naked axis cylinder processes. Some workers have succeeded in staining them by means of the Bielschowsky method for neurofibrils, but in most cases the most satisfactory method will be found to be van Gieson's stain by which the fibers are colored a yellowish brown. They stain red with eosin.

The ganglioneuroma is formed of large pyriform fully formed ganglion cells, very different from the primitive cells of the neuroblastoma. In many cases neurofibrils, beautifully shown by means of the Bielschowsky method, may be demonstrated arising from these cells. Occasionally, as in Dunn's [2] case, areas of neuroblastomatous structure are scattered throughout the ganglioneuroma.

The paraganglioma, composed of chromaffin cells, may arise in the suprarenal medulla, in the carotid gland, and in other situations where chromaffin tissue is found. It is a benign tumor, small in size, and of great rarity. As already remarked, a combination of two types is not uncommon and in exceptional cases all three types have been represented. Wahl's [3] case is a particularly interesting example of this condition, for it contained neuroblastomatous tissue, ganglioneuromatous tissue and chromaffin tissue.

Theoretically, a neuroblastoma may arise wherever neuroblasts—originally derived from the neuro-epithelial canal—are found. In actual practice they most often occur in the medulla of the suprarenal. The abdominal sympathetic and the retina also may be the site of these tumors; in the retina the condition is usually known as glioma or neuro-epithelioma. Any part of the sympathetic system, however, may be involved. Symmers [4] describes a case in which the tumor appeared to originate in the scapular region. This patient was operated on four times; the first two specimens seemed to be ordinary round cell sarcoma, but the third and fourth specimens presented the typical structure of neuroblastoma. Ewing [5] refers to one in which the starting point appeared to be the femoral sympathetic plexus. It is curious to note

2. Dunn, J. L.: Neuroblastoma and Ganglioneuroma of the Suprarenal Body, J. Path. & Bacteriol. **19**:456, 1914.

3. Wahl, H. R.: Neuroblastoma, J. M. Research **30**:205, 1914. A complete bibliography on neuroblastoma will be found in this paper.

4. Symmers, Douglas: A Recurrent Neuroblastoma of the Scapular Region, J. A. M. A. **60**:337 (Feb. 1) 1913.

5. Ewing, J.: Neoplastic Diseases, 1922, p. 774.

that one of the least common sites for a neuroblastoma is the central nervous system; it is the sympathetic and its derivatives in which the tumor is most apt to develop.

Neuroblastoma of the retina is a tumor that has several peculiarities all its own. It is practically confined to infants and young children. Its familial tendency is most remarkable, as many as ten children out of a family of sixteen having died of this disease. Frequently the disease is bilateral. By many workers this tumor is regarded as a glioma. The question cannot be answered definitely one way or the other, for the fibers do not stain characteristically with either the special neuroglia stains or the Bielschowsky method for axis cylinders. Metastases to neighboring lymph nodes and distant organs are late and of rare occurrence, but invasion of the optic nerve and then the brain is common.

A striking feature of the neuroblastoma, especially in comparison with the glioma, the common tumor of the central nervous system, is the early age at which it appears. Of the three cases described in this article the ages were 5 months, 3 years, and 4½ years. In the great majority of cases the disease appears before the age of 5. Dunn's patient was most exceptional in being 14 years of age. The condition has been known to occur in the stillborn. This early age incidence is what one would expect in a neoplasm that appears to arise from primitive nerve cells.

One or frequently both suprarenals may be involved, and many cases previously reported as sarcoma of the suprarenal in infants must now be regarded as neuroblastoma. Two main clinical types have been recognized, the first by Pepper, the second by Robert Hutchison.

Pepper,[6] whose article entitled "A Study of Congenital Sarcoma of the Liver and Suprarenal" appeared in 1901, called attention to the fact that there was a group of cases in infants in which congenital malignant disease of the suprarenal was associated with great enlargement of the liver, the disease being usually, although by no means always, confined to the abdominal cavity. The liver in Pepper's case showed the same extremely diffuse involvement as will be described presently in Case 3, in which the primary tumor was not in the suprarenal but in the retina.

In 1907 Hutchison[7] pointed out that a striking clinical picture in young children (he describes ten cases) was presented by the association of a tumor of the suprarenal with ecchymoses of the eyelids,

6. Pepper, W.: A Study of Congenital Sarcoma of the Liver and Suprarenal, Am. J. M. Sc. **121**:287, 1901.

7. Hutchison, R.: Suprarenal Sarcoma in Children with Metastases in the Skull, Quart. J. Med. **1**:33, 1907.

unilateral exophthalmos and the frequent appearance of tumefaction of the temporal region. The clinical picture of the child with an apparently bruised face, a protruding eye, and lumps over the surface of the cranium, is a most arresting one. It is not only in the cranium that the bony matastases occur, but also in the ribs, vertebrae, sternum and tibia, while in addition there may be involvement of the regional glands, the liver and the lungs.

Much light is thrown on the method of spread of these suprarenal tumors by the work of Frew [8] in 1911. Although the subject of his article is "carcinoma originating in the suprarenal medulla in children," the disease he describes is neuroblastoma. The chief point Frew makes in his paper is that there are two different clinical syndromes and pathologic pictures, depending on which suprarenal is affected. As both suprarenals are often involved this distinction must not be pressed too far, and perhaps the most valuable feature of Frew's contribution is the demonstration of the method of spread. From a study of his own cases and those in the literature he concludes that spread takes place along the lymphatic vessels, and that the clinical and pathologic picture is largely dependent on the anatomic arrangement of these vessels. The lymphatics of the left suprarenal have rich connections with the lumbar glands. In tumors of the left suprarenal he found enlargement of the preaortic group of lumbar glands with extension (1) downward along the aorta, the common iliac, and the internal and external iliac arteries; (2) to the gland at the hilum of the liver and then into the substance of the liver along the portal spaces; (3) up through the posterior mediastinum along the line of the thoracic duct and onward through the anterior deep cervical glands, along the carotid sheath, and through the base of the skull along the internal carotid artery, and (4) along the glands running forward in the intercostal spaces. The bony and lymphatic involvement corresponded closely, and it is easy to understand how the cranium, the superior and inferior maxillae, the ribs, the vertebrae, the sternum, and in one case the iliac bones became the seat of secondary growths. Involvement of the sphenoid results in a marked bulging forward of the posterior wall of the orbit, with inevitable exophthalmos. In six out of eleven of these left-sided cases there were pains in the legs mistaken for rheumatism, and probably due to pressure on the nerves by the enlarged lumbar glands.

In those cases in which the primary growth was in the right suprarenal the path of dissemination was quite different. On the right side the lymphatics from the suprarenal run into the right lymphatic trunk without establishing any extensive connections with the lumbar glands.

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8. Frew: On Carcinoma Originating in the Suprarenal Medulla in Children, Quart. J. Med. 4:123, 1911.

The primary growth is generally much larger than on the left side, and the disease is more often confined to the abdomen. Involvement of the kidney is frequent by direct extension into the pelvis. The glands that may be implicated are those on the upper surface of the liver, the anterior mediastinal glands on the right side, the glands at the root of both lungs, and the anterior deep cervical glands on the right side. The surface of the liver is extensively involved (twenty out of twenty-nine cases) both from the infected glands on its upper surface, and owing to the close apposition of the suprarenal to the bare area of the liver.

We may say, therefore, that in the left-sided cases it is the liver (portal tract), the ribs, the cranium and the thoracic duct and its tributaries (including those from the lower abdomen) which are affected, while in the right-sided cases the upper surface of the liver, both lungs, in a few cases the cranium, and the right lymphatic trunk and its tributaries are involved.

The tumor in the suprarenal, especially when situated on the right side, may attain a great size, even extending down as far as the crest of the ilium. More often, however, it is about the size of a cricket ball. In our own case (Case 1) it was nearly twice the size of the kidney, soft in consistency and hemorrhagic in appearance. Degeneration is common and there may be cyst formation. The glandular swellings are of the same nature as the primary growth. The liver is considerably and sometimes enormously enlarged. In Dunn's patient, a boy, aged 14, it weighed 7,290 gm. In our third case, in which the primary tumor was in the retina, it weighed 1,300 gm., a remarkable figure for a boy hardly 3 years of age. The liver is usually found to be studded with separate tumors, but occasionally there is a diffuse infiltration of tumor cells so extreme in degree that barely a trace of liver tissue remains. This was particularly noted in Pepper's case and in one of Dunn's cases, in both of which the right suprarenal was involved. In Dunn's other case, a neuroganglioma of the left suprarenal, the liver was unaffected.

Our own experience in this respect is not without interest. In the first case, in which the primary tumor was situated in the left suprarenal, there were scattered nodules in the liver. In the second, in which the tumor originated in the abdominal sympathetic on the left side, the liver was quite normal. In the third, in which the primary growth was in the retina, the liver, although extending to below the umbilicus, did not show a single nodule, but was almost entirely replaced by a diffuse infiltration of neuroblastic cells.

The bony lesions, and especially those in the skull, are among the most remarkable features of the disease. The tumor cells reach the bone marrow where they form soft hemorrhagic masses which may

resemble blood cysts rather than solid tumors. This was observed in our first case. In our second case, however, there was a most peculiar invasion of the bones of the cranium, apparently from without, producing a serrated array of bony spicules like the séracs on the ice fall of a glacier (Fig. 5).

Of the three cases reported here, all seen within a few months, the first originated in the suprarenal medulla, the second in the abdominal sympathetic, and the third in the eye.

## REPORT OF CASES

CASE 1.—*History.*—An Italian boy, aged 5 months, was admitted to the Children's Hospital suffering from exophthalmos of both eyes, a small bluish mass on the lateral margin of each supra-orbital ridge, similar nodules over the vertex of the skull, a bruiselike discoloration of the left upper eyelid, a mass in the upper part of the abdomen in the region of the right kidney, and an enlarged liver. The clinical picture was so characteristic that a diagnosis of neuroblastoma of the right suprarenal medulla with metastases in the skull and both orbits was self-evident. The enlargement of the abdomen had been first noticed five weeks before, and was followed shortly afterward by the protrusion of the eyes. For the first three weeks the abdominal enlargement did not make much progress, but during the last two weeks the mass in the abdomen increased tremendously in size. On admission the entire abdomen was markedly distended and very tender to the touch, but a distinct rounded mass could be made out in the region of the right kidney, and the liver was greatly enlarged. The red cells totaled 3,580,000; the leukocytes, 8,600; polymorphonuclears, 53 per cent.; small lymphocytes, 38 per cent.; large mononuclears, 8 per cent., and eosinophils, 1 per cent. After admission to the hospital, the growth of the tumor advanced with startling rapidity, causing the child great agony, from which he was released by death a week later.

*Necropsy.*—The body was that of a very thin and wasted child, with a markedly prominent abdomen and bilateral exophthalmos. Two small bluish masses, about 1 cm. in diameter, were present immediately external to each eye. The heart and lungs were quite normal, the latter showing no secondary growths. The abdominal swelling proved to be due to a greatly enlarged liver. Both the surface and the interior of the liver were studded with very soft hemorrhagic tumors, the largest of which measured only 2 cm. in diameter (Fig. 1). The medulla of the right suprarenal was replaced by a hemorrhagic mass nearly as large again as the kidney, at one side of which a small piece of suprarenal cortex could be recognized. The retroperitoneal glands to the inner side of the right kidney were converted into a mass the size of a hen's egg. The suprarenal medulla on the left side was replaced by a small soft tumor, 2.5 cm. long and 1.5 cm. broad. The kidneys were normal. Another mass of malignant glands, 4 cm. long and 1 cm. broad, lay along the left side of the vertebral column at the level of the third and fourth dorsal vertebrae. On the inner surface of the second rib on the left side there was a chain of hemorrhagic malignant glands, and the interior of the rib contained a small tumor nodule. The most remarkable of all the metastases, however, were those in the skull (Fig. 2). These were of the nature of blood cysts and were so soft and hemorrhagic and so incorporated with the bone that they could not be removed. On the orbital plate of the frontal bone on each side there was a red pulpy mass the size of a cherry, that on the

left being slightly the larger. The sella turcica was occupied by a similar mass as large as a hen's egg. In the middle cranial fossa were four nodules on the right side and three on the left, while on the inner surface of the petrous temporal on the right side there was a much larger mass.

*Microscopic Examination.*—The characteristics of the tumor are best studied in the suprarenal, the liver, and the metastases in the glands. The growths in the cranium were so soft, hemorrhagic and necrotic that they presented no characteristic morphology.

The suprarenal tumor on both sides consisted of a highly cellular tissue, showing profound necrosis in some places and marked hemorrhage in others. The cells of which the tumor was composed were of two main forms (Fig. 3). The

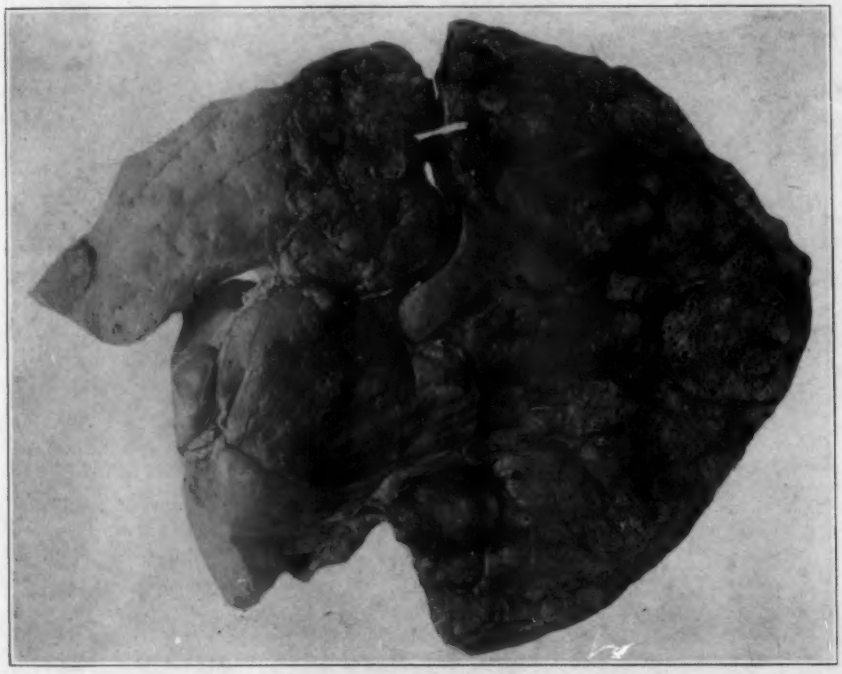

Fig. 1 (Case 1).—Tumor nodules in liver.

first of these was a large, epithelium-like cell, with abundant cytoplasm, vesicular nucleus and a distinct eccentric nucleolus. Although in places the arrangement of these cells may be described as alveolar, for the most part they occur in loose masses; many of the cells showed marked fragmentation of the nucleus. These cells, which are considerably more numerous than the second form, are evidently well differentiated, and they bear some resemblance to the cells forming the chromaffinomatous part of the tumor in Wahl's case. They should, however, merely be regarded as well differentiated neuroblasts.

The other type of cell, which is more primitive in structure, approximates much more closely to the ordinary cell of the neuroblastoma. It is a small cell with scanty, almost invisible cytoplasm, and an intensely hyperchromatic nucleus that occupies almost the entire body of the cell. These cells are for the most

part distributed among the more abundant members of the first group, but in places they are collected into solid masses.

The bundles of fibrils that form so marked a feature of the neuroblastoma are very much in evidence (Fig. 4). Arranged in parallel fashion in fine bundles, many of the fibrils are seen to be nucleated. Nowhere can be seen a trace of the peculiar ball-like structures into which the neurofibrils are so frequently rolled. As a natural sequence there is a complete absence of rosettes, for these are formed by the grouping of the neuroblasts around the little balls of fibers. The fibers, although quite well shown in specimens stained with hematoxylin and eosin, are best demonstrated in van Gieson and iron-hematoxylin preparations, in which they are yellowish brown. They do not stain well with Mallory's phospho-

Fig. 2 (Case 1).—Tumor nodules in skull.

tungstic acid hematoxylin, and refuse to take the stain with Bielschowsky's method.

In the liver the line of demarcation between the tumor and the liver cells is sharply drawn. The tumor cells, which are arranged quite diffusely, are exclusively of the epithelial type; no examples of the neuroblastomatous type are to be seen. The fibrillar element is much in evidence, the fibrils being arranged in longitudinal bundles. The liver cells are the seat of cloudy swelling and fatty degeneration, and many new bile ducts are apparently in process of formation.

The metastases in the lymph glands present a somewhat confusing cytologic picture. The principal cell once more is that of the epithelial type, but many of these show so profound a degree of degeneration involving either karyorrhexis or pyknosis of the nucleus that it is impossible to be certain if cells of the neuroblastomatous type are present. The alveolar arrangement of the cells is

fairly well marked, and there are many bizarre forms, some of which are multinucleated; a few appear to have fused to form syncytial masses. The lymph sinuses are distended by tumor cells.

This case, therefore, must be regarded as one of a neoplasm originating apparently in the medulla of both suprarenals, with metastases in the liver, the lymph glands, the ribs and the cranium, a tumor composed mainly of well differentiated cells, together with small more primitive cells and bundles of neurofibrils, but with an absence of rosettes.

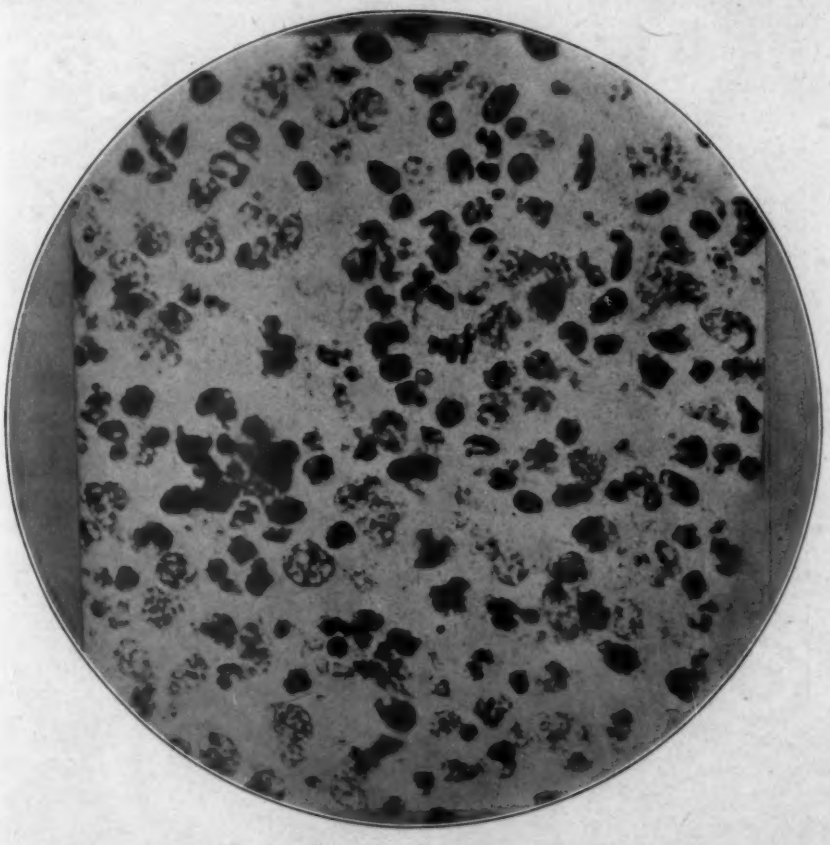

Fig. 3 (Case 1).—Two types of cell.

CASE 2.—*History.*—A boy, aged 4½ years, had as his first symptom proptosis of the left eye. This gradually became more marked, and in the course of three months the right eye began to show the same condition. No tumor could be detected in either eye. A diffuse bruise-like discoloration appeared in both upper lids, and soon the upper part of the cranium could be felt to be covered with soft nodules. No abdominal tumor could be detected. The child suffered great pain. The left eye became blind, terribly protuberant, and finally the globe ruptured. The course of the disease was much slower than in the first case, and the child did not die until five months after the onset of the symptoms. In

spite of the absence of any palpable abdominal mass a diagnosis was made of suprarenal neuroblastoma with metastases in the cranium and orbit.

*Necropsy.*—The head was covered with prominences of very soft consistency, particularly numerous in the frontal region and of varying size, but for the most part about 1 inch (2.5 cm.) in diameter. The face presented a terrible appearance, for both eyes bulged forward horribly and the globes of both were ruptured. When the scalp was removed, it was found that the tumors were very similar

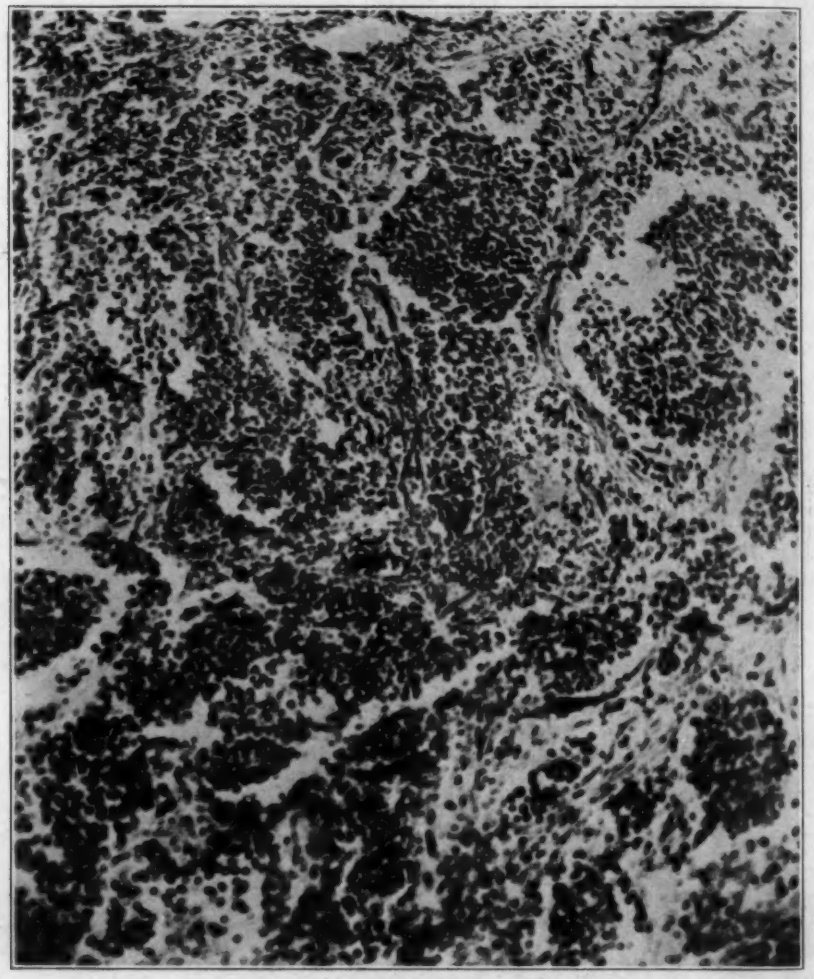

Fig. 4 (Case 1).—Bundles of fibrils between masses of cells.

to those in the first case, being reddish or purple and so extremely soft as to resemble the consistency of leukemic bone marrow. The skull cap was greatly thickened in the anterior part of the temporal region on the left side, measuring 2 cm. in diameter. A thickening that was made up of tissue similar to that which constituted the other tumors was pervaded by fine parallel spicules of bone, which gave it the appearance sometimes seen in an ossifying sarcoma of

bone. When the soft part of the tumor was removed in the septic tank the appearance presented was a most peculiar one (Fig. 5). The little spicules of bone stuck up like the quills of a porcupine, and it was evident that they owed their appearance not merely to destruction of the intervening bone, but to the actual formation of fresh periosteal bone. The roof of the orbit on both sides was perforated by soft masses, and the orbital cavity itself was occupied by large masses of tumor growth. The inner surface of the skull cap as well as the outer showed a remarkable degree of erosion, although the areas on the two surfaces did not correspond with one another. The inner erosions appeared to be caused by the action of several masses of soft, bright red material attached to the surface of the dura mater, although it is more than probable that the tumors originated in the bone rather than in the membranes. The floor of the skull was free from tumor growth. The heart and lungs were normal. The

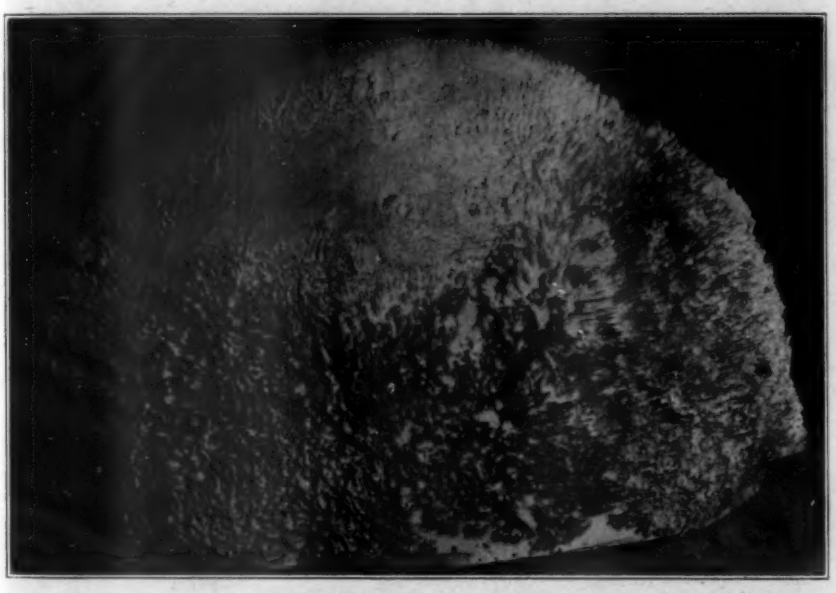

Fig. 5 (Case 2).—Peculiar appearance of cranium.

fifth, sixth and seventh ribs on the left side were covered with large, rounded, soft dark masses, identical in appearance with the tumors in the skull. The left femur contained a similar nodule (Fig. 6).

As the case was regarded as a characteristic suprarenal neuroblastoma, it was both with surprise and chagrin that on opening the abdomen we found the suprarenals to all appearance normal. No tumor formation could be seen in the liver, the kidneys, or any part of the abdominal viscera, but on the left side of the bodies of the second, third and fourth vertebrae an oval mass was discovered 6.5 cm. long and 2.5 cm. wide. This mass was dark in color, firmly attached to the vertebral bodies, and very much denser than the tumors in the ribs and the skull. At first it was thought that this mass was a group of enlarged glands, but subsequent examination proved it to be a tumor arising from the abdominal sympathetic.

*Microscopic Examination.*—The tissues were not fixed for some hours after the postmortem was performed, so that the cytologic picture was not all that

could be desired. The abdominal tumor was composed of cells and bundles of neurofibrils of the same appearance and giving the same staining reactions as those described in the preceding case. Again the fibers were arranged in long bundles, with a few balls but no rosettes (Fig. 7). The cells, however, were very different (Fig. 8). They were nearly all more or less elongated, and some were quite pyriform, so as strongly to resemble ganglion cells. The nucleus was very dense and no structure could be made out, but this may have been because of the delayed fixation. Here and there but only occasionally, there was a clump of small round cells with hyperchromatic nuclei, which might be taken for neuroblasts. At the edge of the section there was a sympathetic ganglion with

Fig. 6 (Case 2).—Nodules in femur and ribs.

normal cells and fibers. The ganglion was partly surrounded by tumor cells and there appeared to be little doubt that the tumor had originated from a neighboring ganglion.

In the ribs the metastases presented a similar structure, but in the cranium the tumor was purely cellular, no fibers being visible. Here, moreover, the tumor had become purely neuroblastomatous in type, the ganglion cells having entirely disappeared.

The left suprarenal presented a condition of great interest. The cortex was perfectly normal, but no trace of the medulla could be found. Its place was taken by fibrous tissue, in which were scattered several groups of large ganglion cells (Fig. 9). These were much more numerous than the occasional ganglion cell seen in the normal suprarenal medulla.

This case must be regarded, then, as a ganglioneuroma originating in the ganglions of the abdominal sympathetic chain on the left side, with metastases in the ribs and cranium and maldevelopment of the suprarenal medulla on the left side.

Case 3.—*History.*—A boy, aged 2 years, was brought to the General Hospital in August, 1922, with a peculiar condition of the right eye. When the eye was

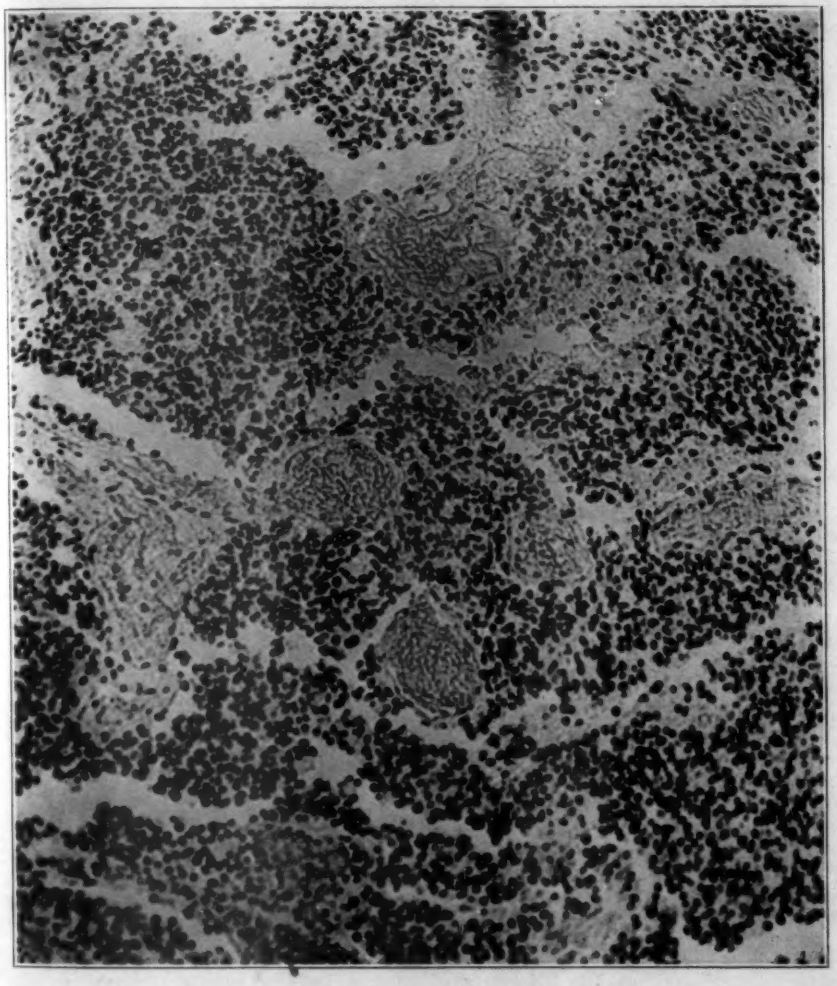

Fig. 7 (Case 2).—Bundles of fibers.

moved from one side to the other it was noted that a white body floated across the pupil, a phenomenon that could easily be observed at a distance of several feet. Ophthalmoscopic examination showed a yellowish-white body floating in the vitreous like a mass of tuberculous sputum. Coursing over it were several immensely dilated veins. The eye was totally blind. Operation was refused, but the child was brought back next July with great pain in the eye, which was

swollen and showed increased tension. Enucleation was performed, and revealed a soft white tumor which not only completely filled the vitreous but invaded the optic nerve (Fig. 10). On account of the latter finding he was submitted to radium and roentgen-ray therapy, but without benefit, for he was readmitted some four months later with a recurrence in the orbit. The tumor grew with terrible speed, and in the course of three months where once the eye had been, there projected a huge spherical mass the size of a tangerine, ulcerated everywhere and bleeding profusely at the slightest touch (Fig. 11). So large was the tumor that it almost closed the nostril on that side. The abdomen gradually became

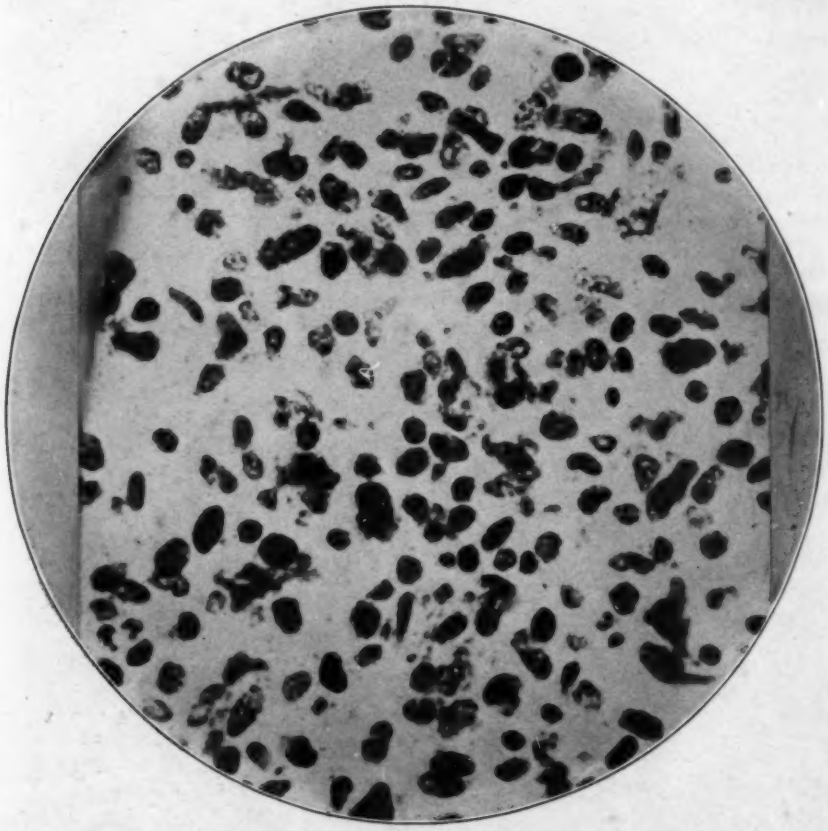

Fig. 8 (Case 2).—Cells composing abdominal tumor.

distended, and it was discovered that this distention was due to marked enlargement of the liver. In the beginning of March, 1924, a nodule appeared in the left parieto-occipital region. This rapidly increased to the size of a walnut; it was extremely soft and tender on pressure. Toward the end slight fever developed, repeated profuse hemorrhages from the orbital tumor produced a condition of great anemia, and the patient died, March 10.

*Necropsy.*—When the abdomen was opened, a huge liver bulged forward. It was bright cherry red, weighed 1,300 gm., and extended for half an inch (1.27 cm.) below the umbilicus. The external surface was perfectly smooth and showed no hint of any tumor nodules. The cut surface was of the same bright red, although

mottled with yellow areas, and was again perfectly free from any suggestion of tumor formation. The other thoracic and abdominal organs were normal. The entire sympathetic chain and the suprarenal glands were examined with great care, but no lesion was found. When the abdominal organs were removed a chain of bright red lymphatic glands was seen along the lower part of the abdominal aorta and extending for a short distance along the common iliac and

Fig. 9 (Case 2).—Fibrosed suprarenal medulla, showing ganglion cells.

internal iliac arteries. They were present on both sides of the aorta, but were rather more numerous on the right. Most of them were about the size of a pea, but a few were two or three times that size. It is of great interest to note that both Frew and Symmers mention that the abdominal lymph glands in their cases were of a peculiar bright red.

The surface of the cranium presented numerous rounded elevations, dark blue, soft in consistency and about the size of a cherry. When the skull cap was

held up to the light it was seen that there were large numbers of small nodules of a similar character in the substance of the bone. Many of these were only 5 mm. in diameter. A few of the large nodules appeared on the inner surface of the bone, but the membranes were not invaded. The right orbit was occupied by a large soft tumor the size of a tangerine, a large part of which projected on the surface. The tumor had perforated the roof of the orbit, infiltrated the dura mater, and extended over the floor of the skull in front of the sella turcica into the anterior cranial fossa on the left side. The brain and the left eye were quite normal.

Fig. 10 (Case 1).—Primary tumor of eye.

Fig. 11 (Case 1).—Tumor that developed in orbit after removal of eye.

*Microscopic Examination.*—The tumor of the eye presented a picture quite characteristic of neuroblastoma of the retina or retinal neuro-epithelioma. It is true that no rosette bodies nor glandlike spaces were to be seen, but the perivascular or perithelial arrangement of the cells so often seen in these tumors was well shown (Fig. 12). The many thin-walled blood vessels were surrounded by a mantle of dark cells with scanty cytoplasm and hyperchromatic nucleus. Beyond this mantle an ever increasing number of cells showed marked karyorrhexis and still further out there was complete necrosis with loss of all structure. The cells nearest the vessel were much elongated, approximating to the rod type of cell sometimes seen in these tumors, and fine processes proceeded from them to establish connections with the wall of the vessel. Mitotic figures were numerous.

Examination of the large orbital tumor and of the nodules in the skull was disappointing. Here the necrosis was so profound that no definite structure or arrangement could be made out.

The liver presented a remarkable appearance. Although to the naked eye no tumors were visible, microscopic examination showed that the organ was in reality one great diffuse tumor. It is no exaggeration to say that the liver was almost entirely composed of tumor cells, with only here and there a few groups of degenerated liver cells. How life could be maintained with such a minimum of hepatic tissue must remain a mystery. The cells were perfectly diffuse in their arrangement and showed no attempt at rosette formation; no fibrils could be made out, and the general appearance was that of a round cell sarcoma.

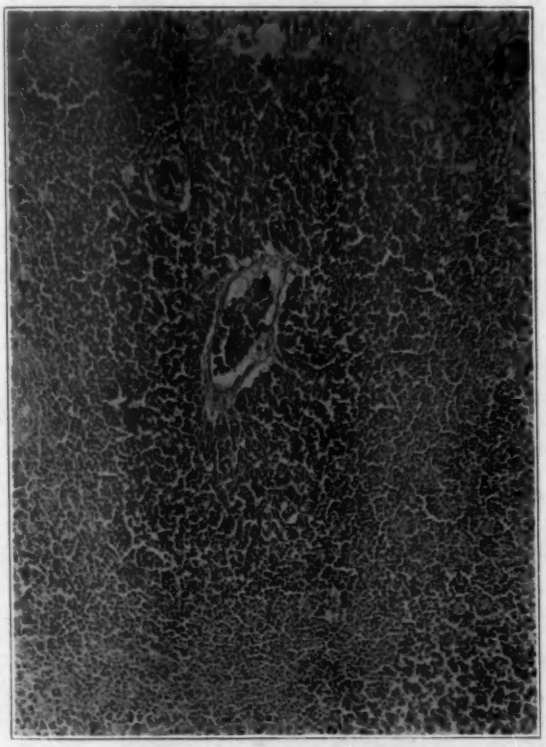

Fig. 12 (Case 3).—Perivascular arrangement of cells in retinal tumor.

This is one of those somewhat uncommon examples of neuroblastoma or neuro-epithelioma of the retina in which metastases are set up in the internal viscera.

## SUMMARY

In three cases of tumors occurring in children, one tumor arose from the suprarenal medulla, the second from the abdominal sympathetic, and the third from the retina.

These three tumors, although originating from different organs and differing in their microscopic structure, nevertheless possess features

in common which justify their common consideration. They may all be regarded as developmental tumors arising in children from the neuroblast at different stages of its development. In the first two the spread was apparently by the lymphatics, in the third by the blood stream. In all the most striking metastases were in the cranium.

# FRACTURES OF THE HEAD OF THE FEMUR

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CHICAGO

Fractures of the head of the femur are exceedingly rare. The first case to be reported in the literature was in 1869 by Birkett.[1] Since then thirteen other cases have been reported. In at least four of these the pathology is somewhat doubtful. I add the fifteenth case and an analysis of the series.

## REPORT OF CASE

Miss H. B., aged 30, admitted to Evanston Hospital, Evanston, Ill., Nov. 1, 1924, had been injured while riding in an automobile two days before. The machine crashed head on into a telegraph pole. With the impact, the patient's knee or upper part of the leg was driven forcibly into the front of the car, probably with the thigh slightly abducted. The physician who was called, after examining the patient and measuring her legs, told her that she had strained a muscle. He permitted her to go to the bathroom, but advised her to remain in bed at other times and to be careful of her hip. After two days of suffering, another physician was called and he referred the patient to me.

On admission to the hospital, the right thigh was flexed and adducted. Extension was possible to some 150 degrees, but abduction was impossible. There was an abrasion and contusion of the proximal third of the anterior surface of the leg. Practically all movements of the thigh were extremely painful. A roentgen-ray examination was made immediately and showed the femur to be dislocated upward, outward and backward. The lower half of the head of the femur was broken off from the remainder of the head and remained in the acetabular cavity (Figs. 1 and 2).

The patient was completely anesthetized with ether and the dislocation was reduced. This was effected by, first, extreme flexion of the thigh, then extension to about 110 degrees, and then slow, steady abduction against considerable resistance. The fractured head slipped into the acetabulum with a crepitant sound. The motion in the joint at once became free. A roentgenogram taken at this time showed the dislocation to be reduced and the fragments to be in excellent position (Fig. 3).

The leg was suspended for a few days in a Thomas splint used as a cradle, and then was permitted merely to rest on pillows in the bed. Passive and active motion was instituted very early. On the eighteenth day after reduction, the patient was able to walk with the aid of crutches, and on the thirtieth day was able to walk alone.

A serious impediment to walking lay in the presence of a foot-drop. It became a nice question to decide whether or not this extensor paralysis came from an injury to the sciatic nerve either before or during the reduction, or from a contusion to the anterior tibial nerve. Accordingly, Dr. Lewis J.

1. Birkett, John: Tr. Med. Chir., London **52**:133, 1869.

Pollock was called in consultation and made the following report: "There is paralysis of the tibialis anticus and probably paralysis of the extensor communis digitorum; occasionally there seems to be some contraction of both muscles. The peronei can be moved. The gastrocnemius and tibialis posticus are normal. There is anesthesia over the distribution of the external popliteal, but no analgesia. All the muscles react to faradism, the tibialis anticus very little, the others more. In my opinion, there is contusion of the external popliteal nerve, a dissociated lesion which is spontaneously recoverable." He recommended faradic stimulation of the paralyzed muscles, and this was instituted and continued until the discharge, at which time the return of extensor function was about 30 per cent.

On discharge, fifty-three days after admission, the patient was able to walk a mile in a day. Owing to the slight foot-drop still present, she walked with a slight limp and there was a compensatory tilting of the pelvis. After use there was some aching of the entire leg, but this symptom has steadily decreased. After discharge the patient left for her home in a distant city. Letters received from time to time indicate complete recovery as far as the hip is concerned, and steady progress in the alleviation of the foot-drop.

May 15, 1925, the patient reported that she could easily walk four miles at a time without limping. She is able to extend the right foot practically as well as the left, has gained 20 pounds (9 kg.) in weight, and is in excellent health and spirits.

The cases from the literature are here reported in abstract:

Birkett,[1] in 1869, reported a case of a woman, aged 35, who fell 25 feet (762 cm.) from a second floor window to the pavement, sustaining a fracture of the head of the femur, a posterior dislocation of the femur backward and upward, a fracture of the skull and laceration of the brain. She died a few minutes after admission to the hospital. Necropsy revealed the fracture to be very even and on a plane with the vertical axis of the femur. The fragment of the head, to which the greater portion of the ligamentum teres was attached, remained in the acetabulum. In Birkett's opinion, either the head was split by being driven against the strong edge of the acetabulum, or the strength of the ligamentum teres was sufficient to overpower the cohesion of the bony tissue, while the femur was driven upward and backward.

Moxon,[2] in 1872, reported the case of a railway porter, aged 30, who was injured by a moving train and died shortly after admission to Guy's Hospital. On examination the position of the limb was that of a dislocation on the dorsum of the ilium. A large dent in the skin corresponded to the junction of the left sacrosciatic ligament with the tuber ischii. Necropsy revealed a portion of the head of the femur in the socket attached by the round ligament. In addition to the fracture there was also a posterior dislocation of the hip.

Riedel,[3] in 1885, described a fracture of the head of the femur in a boy of 15 who had been run over by a heavy cart. The symptoms presented were 5 cm. of shortening, flexion and inward rotation, and crepitation. In addition to this fracture, there were also a dorsal dislocation and a fracture of the acetabulum. For the first three months, the patient received no care. At operation there was found an oblique fracture of the head of the femur, the line of fracture running down into the neck. This fragment had the ligamentum teres attached to

2. Moxon: M. Times & Gaz. **1**:96, 1872.
3. Riedel: XIV Tagung, Deutsche Gesell. f. Chir., 1885.

it. There was a transverse fracture of the balance of the head and both fragments were out of the acetabulum (Fig. 4). The two fragments were removed and the balance of the head reduced into the acetabulum. Three months after operation, there was an ankylosis of the femur and 2 cm. of shortening.

Stimson's[4] patient, reported in 1889, fell through a hatchway and died ten days after injury. At necropsy the dislocated head of the femur was impinged on an exostosis at the rim at the acetabulum. On the anterior surface of the

Fig. 1 (author's case).—Fracture of head of femur (schematic) with fragment remaining in the acetabulum, occasioned by violence and accompanied by posterior dislocation of the hip.

head of the femur was a groove or contusion parallel with and close to the edge of the articular cartilage, an inch (2.5 cm.) long, half an inch wide and an eighth of an inch deep, along which the cancellous structure of the bone was exposed. There also was a dorsal dislocation of the hip.

4. Stimson, L. A.: New York M. J. **50**:163 (Aug. 10) 1889.

Crile,[5] in 1891, reported the case of a man, aged 53, who fell 30 feet from the mast of a vessel into the hold. He died the third day after admission into the hospital. He had a dorsal dislocation of the femur, a fracture of the base of the skull, a fracture of eight ribs and a fracture of the acetabulum. He was treated for shock; a decompression was performed, and the dislocation reduced. At necropsy one third of the lower part of the head at the junction

Fig. 2 (author's case).—Fragment of head of femur lying tilted in acetabulum before reduction of the dislocation.

with the neck was broken off and attached to the neck by a small ligamentous band, the fragment lying outside the acetabular cavity (Fig. 5).

Braun's[6] case, reported in 1891, was that of a man, aged 37, who was struck by a train and thrown to the side, sustaining a fracture of the head of the femur,

5. Crile, G. W.: Ann. Surg., May, 1891, p. 373.
6. Braun: Arch. f. klin. Chir., 1891, p. 109.

a dorsal (ischiadic) dislocation of the femur, a fracture of ten ribs and a hemorrhage into the pleural cavity. The dislocation was reduced, but the patient died nine hours after admission into the hospital. Necropsy revealed the line of fracture of the head extending from the middle of the ligamentum teres downward and inward to end in the region of the capsule insertion (Fig. 6). In his opinion, the fracture was caused by the sudden dislocation of the head of the femur posteriorly and the pull of the strong ligamentum teres.

Fig. 3 (author's case).—After reduction of the dislocation, both fragments are believed to be in the cavity of the acetabulum.

Roberts,[7] in 1896, reported the case of a man, aged 25, who was caught between bumpers while coupling railroad cars. He sustained fractures of the head of the femur, neck of the femur and of the acetabulum, and a dorsal dislocation of the femur. He was treated for three months, partly by traction,

7. Roberts, J. B.: Ann. Surg. **23**:207, 1896.

before consulting Roberts, who attempted reduction under ether with pulleys, which nearly resulted fatally. Roberts then performed an open operation to reduce the dislocation, but the patient died immediately afterward. A partial necropsy was performed. The specimen removed consisted of the upper end of the femur and part of the neck, and two portions of the femoral head (which had been cut by the chain saw in the operation), to which portions of the ilium were attached. The head was evidently split at the time of fracture for there was a piece of bone united at one side of the head in an irregular manner. The head had probably been burst asunder by the neck being driven into it.

Durant and Destot [8] reported three cases in 1904. The first patient was a man who fell from a considerable height. Roentgen-ray examination revealed a fracture of the head of the femur. This was treated by massage, heat and mobilization. The result was a partial and painful ankylosis.

The second case [8] was that of a man who fell backward. Roentgen-ray examination revealed a fracture of the head of the femur. The treatment employed was massage, heat and mobilization and the result, a partial and painful ankylosis.

The third case [8] was that of a man of 44, who presented symptoms of a luxation. No history of the accident is given. Roentgen-ray examination revealed a fracture of the head of the femur and a posterior dislocation of the hip. This patient also was treated by massage, heat and mobilization after the dislocation was reduced. The result was a partial and painful ankylosis.

Moravek,[9] in 1912, reported the case of a man, aged 58, who fell, striking the anterior surface of the hip on a sharp iron wedge. For seven years preceding the examination by Dr. Moravek, the patient had had pain on the anterior surface of the thigh and abdomen and limitation of motion. Roentgen-ray examination revealed an isolated fragment of the outer border of the head of the femur at the site where this becomes the neck of the femur. The patient was kept in bed for six weeks with no other treatment, and made only an incomplete recovery.

Satta [10] in 1922, reported the case of a man, aged 36, who was injured in a motorcycle accident in which the machine fell on the patient. There was 6 cm. of shortening and adduction of the thigh; the trochanter was above the Roser-Nélaton line, and there was limited passive motion. There was no active motion. Roentgen-ray examination revealed the head of the femur fractured downward and backward. The fragment remaining in the acetabulum included the attachment of the ligamentum teres. About half the head was in the acetabulum and appeared as though it had been cut by a saw (Figs. 7 and 8). There was a posterior dislocation of the femur. Reduction under anesthesia had been attempted twice without success before the patient's admission to the hospital. Operation was performed on the fifth day. The dislocation was reduced on a traction table, but could not be maintained. Therefore, the fragment of the head was removed from the acetabulum, the remainder of the head shaped, covered with fascia lata and placed in the acetabulum. On the nineteenth postoperative day, the head was found out of the socket. On the

8. Durand and Destot: Bull. Soc. de. chir. de Lyon **6**:376, 1903; Lyon méd. **102**:212, 1904.

9. Moravek, A.: Cas. lek. cesk., 1912, No. 36, p. 1105; abstr., Centralbl. f. Chir., 1912, No. 47.

10. Satta, F.: Chir. d. org. di movimento **6**:97 (Feb.) 1922.

twenty-fifth day, a second arthrotomy was performed. The patient had a stormy convalescence, developing erysipelas and abscesses on the fifty-second day. He finally recovered with a total ankylosis of the hip joint.

Hinsdale,[11] in an article appearing in 1923, reported the case of a woman, aged 55, who fell in attempting to jump from a motor boat to a wharf. She tripped over the gunwale of the boat and fell on the wharf, striking her left hip. The thigh was partly flexed; the foot was inverted; crepitus was present and some shortening. Roentgen-ray examination revealed a fracture of the

Fig. 4 (Riedel's case).—Oblique and transverse fractures of head of femur (schematic), occasioned by violence and accompanied by posterior dislocation of hip.

head of the femur with the fragment lying on the lower margin of the acetabulum. The fragment was 15 by 35 by 30 mm.—one third of the head of the femur—and included the insertion of the ligamentum teres. There was a posterior dislocation of the femur dorsally. An ineffectual attempt was made

11. Hinsdale, G.: Fracture of Head of Femur, with Dislocation on Dorsum of Ilium, J. A. M. A. **80**:469 (Feb. 17) 1923.

to reduce the dislocation under ether. Open operation was then performed, the fragment removed and the dislocation reduced. Extension was maintained for three weeks. The patient was able to walk in two months and was perfectly well in four months.

Levin [12] reported a case in 1923 of a man, aged 33, who was struck by a blow from a truck. He continued to work but walked with a limp into the hospital on the next day because of the pain. There was no swelling or shortening. There was some tenderness on pressure over the ilium. Roentgen-ray examination was negative. The treatment instituted was rest in bed. On

Fig. 5 (Crile's case).—Lower third of head of femur (schematic) broken off and lying outside cavity of acetabulum, occasioned by fall of 30 feet and accompanied by posterior dislocation of hip.

the forty-seventh day, he developed a temperature of 101.6 and a septic or scorbutic condition of the mouth. An exploratory puncture of the hip joint was performed on that day and repeated four days later, being negative on both occasions. Movement was completely limited except under anesthesia. Arthrotomy of the hip joint was then performed but no pus obtained. Traction was applied. On the fifty-fifth day, pus was obtained from the wound. The patient died on the sixty-third day from sepsis in the hip joint and buttock.

12. Levin, J. J.: Brit. J. Surg. **11**:388 (Oct.) 1923.

Necropsy was performed. The head of the femur was found in the acetabulum, separated from the neck and shaft; in fact, there was what Levin considered an ununited fracture of the head of the femur. The joint was septic and contained green, foul smelling pus. The separation of the head from the neck was at a higher level than the epiphyseal line, although posteriorly it did for a short distance coincide with it (Fig. 9).

Fig. 6 (Braun's case).—Fracture of head of femur (schematic), caused by violence and accompanied by posterior dislocation of the hip. The line of the fracture runs through the ligamentum teres.

In addition to the cases referred to above, Cotton[13] states that he has seen "three cases of impaction within the limits of the head of the femur." Fractures of the head of the femur must be distinguished as

13. Cotton, F. J.: Dislocations and Joint Fractures, Philadelphia, W. B. Saunders Company, 1924, p. 506.

a separate entity from the displacements of the upper epiphyses of the femur, which have been so well described recently by Wilson.[14]

In the comparative study of the reported cases it seems wiser to omit certain cases. In Stimson's case, there was not a complete fracture of the head but merely an indentation in it occasioned by contact with an exostosis on the rim of the acetabulum. Robert's case, while strictly

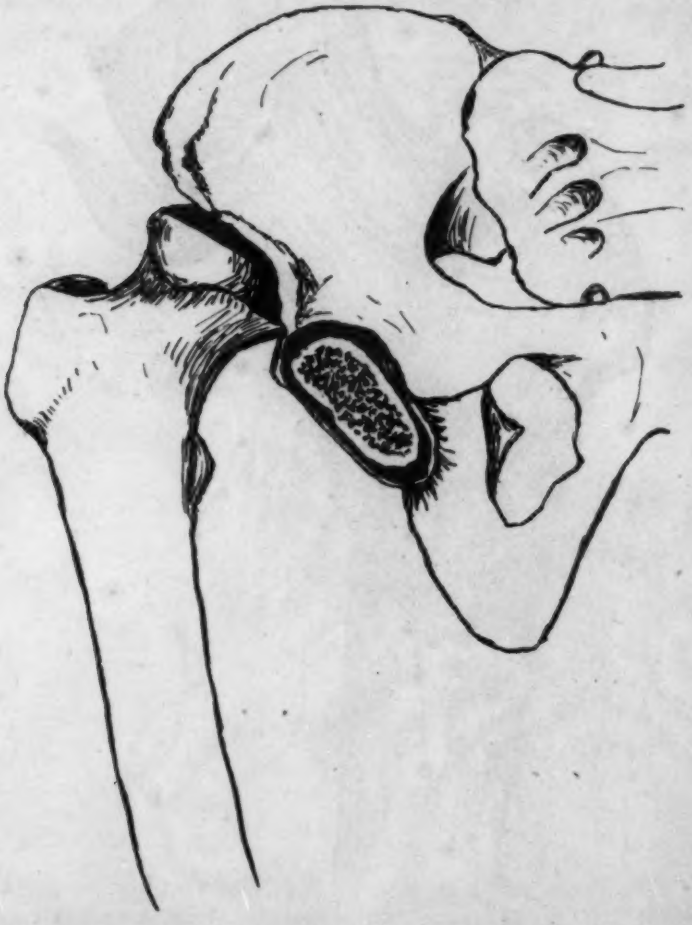

Fig. 7 (Satta's case).—Fracture of head of femur (schematic), occasioned by extreme violence and accompanied by posterior dislocation of hip. The fragment remaining in the acetabulum includes the attachment of the ligamentum teres.

speaking a fracture of the head, seems more logically to be classified as an aberrant type of central dislocation of the femur. Some doubt is thrown on Durand and Destot's first two cases as detailed case reports

14. Wilson, P. D.: Displacement of Upper Epiphysis of Femur Treated by Open Reduction, J. A. M. A. **83**:1749 (Nov. 29) 1924.

are missing, and there were no luxations of the hip present. A sufficiently detailed account of Moravek's case was not obtained to make it of value for study. Levin's case is extremely interesting, but it is rather difficult to include this as a typical case of fracture of the head of the femur despite the necropsy findings.

Accordingly, analysis must be based on nine cases—those of Birkett, Moxon, Riedel, Crile, Braun, Durand and Destot (third case), Satta, Hinsdale and Christopher. Of these nine cases six were in males and three in females. The ages varied from 15 to 55 years and averaged

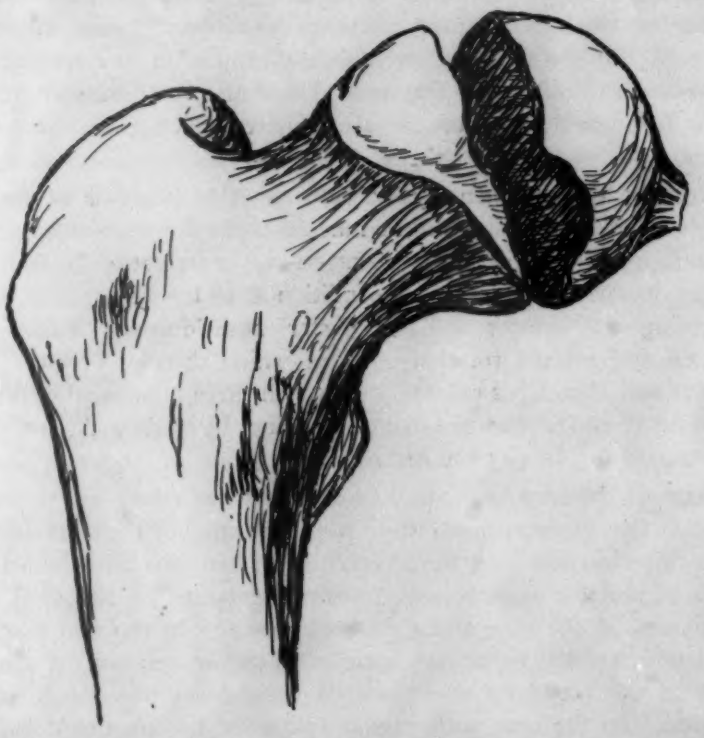

Fig. 8 (Levin's case).—Ununited fracture of head of femur (schematic); necropsy findings in septic joint of scorbutic patient.

37 years. The cause of the accident was extreme violence, such as train or automobile accidents or falls from a height of 25 or 30 feet in at least seven cases. In Hinsdale's case only was the traumatism less violent. His patient, a woman, aged 55, fell in attempting to jump from a motor boat to a wharf; she tripped over the gunwale of the boat and fell heavily on the wharf, striking her hip.

In all these nine cases there was a posterior dislocation of the hip. The chief factor in the etiologic mechanism of these fractures is the application of a violent force acting either in the long axis of the femur

and toward its proximal end or directly on the great trochanter. This force is suddenly applied during the progress of the posterior luxation so that the sphere of the head of the femur impinges violently against the posterior rim of the acetabulum. This force may be a part of the force which causes the posterior dislocation. In all probability it is always applied when the femur is flexed and slightly adducted. A second factor is the pull of the ligamentum teres which is exerted during the dislocation. Birkett [1] thought that the strength of this ligament was sufficient to overcome the cohesion of the bony tissue while the femur was driven upward and backward. This view is scarcely tenable when it is considered that the ligamentum teres is not strong and ruptures at 14 kg.[13] Stimson's case, in which the ligamentum teres ruptured and the head was incompletely fractured should also be considered. A third factor is probably that of the atmosphere, which exerts pressure to maintain the head in the socket.

Prior to the roentgen-ray examination, the diagnosis in the living was well nigh impossible. In the nine cases the diagnosis was confirmed by necropsy in four cases, by roentgen-ray examination in four cases, and by operation in one. This condition is to be suspected in patients presenting the symptoms of a posterior dislocation of the femur (flexion and inward rotation) and in which there is crepitus (as in Riedel's and Hinsdale's cases), or in which there is unusual difficulty of reduction (Satta). Positive diagnosis cannot be made without roentgen-ray examination, biopsy and necropsy.

Four of the nine patients died shortly after admission to the hospital. In two of these cases, there were fractures of the skull and other severe injuries and in a third, fracture of ten ribs with hemothorax. Three of the five patients who recovered retained an ankylosis of the hip, partial in one case and complete in two. In the two cases with complete ankylosis, there had been an operative removal of the fragments of the fractured head; one of these cases was complicated by infection. In the case with partial ankylosis, the treatment had been limited to a reduction of the dislocation, massage, heat and mobilization (Durand and Destot, third case).

Two patients recovered completely. In one of these (Hinsdale's case), there was an operative removal of the fragment of the fractured head and immobilization in extension for three weeks. In the other case, my own, the treatment consisted of reduction of the dislocation under ether and early immobilization.

It will be seen from the foregoing account that the prognosis as to function is extremely grave in these cases; only two of the nine cases recovered and only one of these (mine) without operation.

SUMMARY

1. Fractures of the head of the femur are exceedingly rare.

2. The diagnosis should be made by roentgen-ray examination.

3. A posterior dislocation of the femur accompanied all the classical cases. The chief etiologic factor is undoubtedly the impact of the dislocating head of the femur against the posterior rim of the acetabulum.

4. The prognosis as to function is grave.

5. The treatment of choice is closed reduction of the dislocation under ether and early active and passive mobilization.

6. Open reduction of the dislocation is necessary in those cases in which attempts at closed reduction have failed. In these cases, the operative removal of the loose fragment or fragments of the fractured head is advisable.

7. In those cases in which closed reduction has been accomplished but in which function becomes progressively worse, operative removal of the fragments is indicated.

8. A small number of cases are mentioned in the literature which may be fractures of the head of the femur *without* posterior dislocation (Durand and Destot, Levin).

# TETANUS

## ITS INCIDENCE AND TREATMENT *

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### THE INCIDENCE OF TETANUS

The present inquiry has been instigated by the occurrence of a case in which tetanus developed and proved fatal in an entirely unsuspected industrial accident; unsuspected both from the circumstances of the injury and the nature of the wound itself. Reference to government reports shows that in the U. S. Registration area the number of deaths per year from tetanus is about 1,500. Not a large figure perhaps but too large for a preventable disease and one for which we have a presumably specific antitoxin. The principal points to be inquired into seemed to be: (1) How much of a burden is this disease to industry? (2) Can definite plans be developed for more systematic prophylactic injections of antitetanic serum? (3) Can the present very unsatisfactory plans of treatment be improved?

In order to get information from widespread sources, 2,000 questionnaires were sent to the entire membership of the Association of Industrial Physicians and Surgeons and to the Railway Chief Surgeons. Replies numbered 584. These replies together with an exhaustive review of English and American literature are the basis of this study.

### IMPORTANCE OF TETANUS IN INDUSTRY

Of the 584 men returning questionnaires, nearly all of whom have extensive industrial and traumatic experience, 365, or nearly two-thirds, stated that they had seen no tetanus in the last four years (1921-1924) in industrial cases. It has been impossible to estimate the number of accidental wounds covered by these men, but in the four years there must have been several millions. The 219 men who had seen tetanus reported less than 250 industrial cases seen in the last four years and 326 nonindustrial cases.

Therefore, 584 men in railroad or industrial work, representing millions of injuries in the last four years, report 250 cases. Economically the disease would not seem important to industry in general. The

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* Read in part before the American Surgical Association, May 6, 1925; in part before the Association of Industrial Physicians and Surgeons, May 26, 1925.

one or two accident insurance companies who took interest enough in the subject to reply to letters do not think that tetanus presents any importance to them.

Other information as to the connection between tetanus and industry is found in Table 1, in which 760 cases from the literature or personal reports are tabulated according to occupation. In this table it will be seen that cases arising in uncontrollable circumstances, such as street, farms, homes, gardens, stables, etc., make up 80 per cent. of the entire incidence of tetanus. Only 20 per cent. arise in conditions now ordinarily within the care of industrial medicine, that is railroads, mills, mines and factories.

*Steel Industry.*—Dr. W. O'Neill Sherman of the Carnegie Company states that in the last fifteen years his company has had approximately 300,000 open wounds and that only two of them developed tetanus. He reports no cases in 90,000 open wounds in the last four years. Dr. L. A. Shoudy of the Bethlehem Steel Works at Bethlehem reports no tetanus in the last four years in 42,000 wounds. Dr. J. H. Lewis of the Bethlehem Company at Buffalo, N. Y., reports one case in approximately 150,000 accident wounds in the last twenty years.

We have the following additional reports from steel plants: Dr. H. E. McGuire of the Jones and Laughlin Company and the Oliver Iron and Steel Company has had no cases (28,000 employes); Dr. J. E. Eichi of the Lockport Steel and Iron Company, no cases in last four years, in 16,000 wounds; Dr. James Jefferson, Bethlehem Plant at Johnstown, one case in twenty years; Dr. C. H. Stone, Bethlehem Company at Coatsville, Pa., no cases in last four years, in 8,000 accidental wounds; Dr. L. G. Harney, American Steel Company, St. Louis, no case in five years; Dr. C. W. Page, Carnegie Company, McKees Rocks, Pa., none in four years; Dr. A. W. Colcord, Carnegie Steel Company at Clairton, Pa., no cases in last four years, in 8,000 injuries; Dr. Sidney McCurdy, Youngstown Steel and Tube Company, one case in last four years, in 17,000 open wounds; Dr. W. H. Means, Bethlehem Company at Lebanon, Pa., no case in seven years, in approximately 30,000 wounds; Dr. R. R. Cummings, Midvale Steel Company, Philadelphia, only one case in twenty years; Dr. W. E. Hodgson, Pittsburgh Steel Foundry Company, none in four years, in 1,500 cases; Dr. A. S. McKinley, Colonial Steel Company, no cases in four years; Dr. L. S. Brookhart, American Steel and Wire Company, no cases in four years; Dr. A. L. L. Baker, Replogle Steel Company, none in four years; Dr. R. C. Engel, McKinley Steel Company, no cases in four years; Dr. R. T. Shipley, Union Alloy Steel Company, no cases in four years, in 72,000 wounds; Dr. L. A. Will, Scullin Steel Company, none in fourteen years, in about 50,000 wounds; Dr. H. S. Murat, American Rolling Mill Company,

none in fifteen years, in about 6,000 wounds; Dr. J. R. Bieler, Canton Steel Company, no case in four years, in "many thousand wounds"; Dr. D. E. Sloan, Pittsburgh Steel Company, Charleroi, Pa., no cases in four years, in about 4,000 wounds; Dr. J. A. Helfrich, Pittsburgh Crucible Steel Company, no cases in four years, in about 10,000 wounds; Dr. F. W. Black, Standard Steel Works, none in four years, in about 4,000 wounds.

*Railroads.*—I have reports from fifty-seven chief surgeons of railroads which indicate almost the same freedom from tetanus in accidental wounds as is shown in steel plants. These fifty-seven chief surgeons report only twenty-nine cases of tetanus in the last four years, though

TABLE 1.—*Classification According to Industry and Occupation*

| | | |
|---|---|---|
| Street | | 159 |
| Farm | | 155 |
| Home | | 100 |
| Stable | | 61 |
| Railroad | | 65 |
| Garden and soil, unspecified | | 40 |
| Mills | | 28 |
| Steel | 13 | |
| Cotton | 6 | |
| Other textiles | 4 | |
| Grist | 2 | |
| Others | 3 | |
| Coal mines | | 23 |
| Machine shops | | 7 |
| Horse shoeing | | 7 |
| Bathing | | 11 |
| Animal industries | | 17 |
| Tannery | 7 | |
| Horsehair plant | 1 | |
| Butcher | 6 | |
| Packing house | 3 | |
| Building trades | | 9 |
| Lumber trades | | 28 |
| Saw mill | 4 | |
| Lumber yard | 4 | |
| Carpentry | 20 | |
| Factories* | | 22 |
| Miscellaneous† | | 28 |
| Total | | 760 |

* Five automobile, three fertilizer, two paper, two car, one each glue, furniture, tire, box, chemical, gelatin, boiler, shoe, foundry and piano.

† Four bakers, four clerks, three printery workers, three shipyard workers, two granite workers, two longshoremen, and one each oyster plant worker, scavenger, express handler, sailor, grocer, well digger, brick-yard worker, felt worker, roofer and engraver.

the total number of wounds seen or reported to them in this period must again be well up in the millions. Forty-five of the chief surgeons report no cases at all in the last four years. Several report no cases in "many years." Five large railroads report only one case each. Nine of twenty-nine railroad cases were in Mexico, Texas and Oklahoma. Railroads having only one case in four years were: Nickel Plate, Northwestern, Southern Pacific, Missouri and Illinois (Missouri) and the Illinois Central. Railroads having no case in four years were: Great Northern, Akron, Canton and Youngstown, Ohio, Spokane International, American Oiler, Baltimore and Ohio, Delaware, Lackawanna

and Western, San Francisco, Burlington, Northern Pacific, Chicago and Eastern Illinois (Chicago), Norfolk and Western, Pittsburgh, Lisbon and Western, Georgia and Florida, Canadian National, Central of Georgia (Mississippi), Pere Marquette, Colorado and Southern, New York Central (Ohio Central Lines), Rock Island, Virginian, Southern Pacific, Chicago, Peoria and St. Louis, Norfolk Southern (Virginia), Pittsburgh and Lake Erie (Pennsylvania), Lake Erie and Eastern (Pennsylvania), Maryland and Pennsylvania (Maryland), Buffalo, Rochester and Pittsburgh, Rutland, Ozark Southern (North Carolina), Mobile and Ohio (Alabama), Atlanta and W. P., Western of Alabama, Georgia Railroad, New York Central (Chicago), Arizona Eastern (Arizona), Bangor and Aroostook, Alton, Boston and Maine, New Mexico Central, Atlanta, Birmingham and Atlantic (Georgia), Spokane, Portland and Seattle (Oregon), Midland Valley (Oklahoma), Tennessee Central and Western Pacific.

*Coal Mining.*—I have reports from chief surgeons of ten large coal mining companies and from eighteen local surgeons to other companies. Dr. W. G. Fulton, Hudson Coal Company, reports one case, that of a man dragged in a mule yard, in 1904 and no cases since in approximately 100,000 wounds. Dr. J. H. Young, Lehigh Coal and Navigation Company, reports no cases in eight years. Dr. C. H. Warner, Susquehanna Coal Company, Dr. H. A. Smith, Lehigh & Wilkes-Barre Company, and Dr. S. P. Mengel, Lehigh Valley Coal Company, report no cases in four years; Dr. F. F. Arndt, Pennsylvania Coal Company, one case in nineteen years; Dr. James Ward, Conshocken Coal Company, no cases in twelve years. In my own experience of twenty-five years with the Glen Alden Coal Company and its predecessor, with an enormous number of wounds we have had seven cases.

The eighteen local surgeons to other companies report between them seven cases in the last four years. In many cases a negative experience dates back much longer.

*Packing Industry.*—Dr. V. S. Cheney of the Armour and Morris Companies at Chicago reports two cases in the last four years in 29,000 wounds. Dr. R. H. Wald, Armour Company at St. Paul, reports one case in four years in 20,000 wounds; Dr. W. R. Addeson, Davis Company, Toronto, no cases in four years, in 14,000 wounds; Dr. W. H. Lipman, Swift and Company, three cases in twenty years, in about 140,000 wounds; Dr. B. F. Buzby, Campbell Soup Company, Camden, N. J., no cases in the history of the company, in about 12,000 wounds a year.

*Miscellaneous Industries.*—Dr. A. K. Smith, medical director of the DuPont Company, reports no cases in four years; Dr. C. H. Wilson, medical director of the American Telephone and Telegraph Company,

does not recall any cases; Dr. J. D. Stark, Hammermill Paper Company, no cases in the last twenty-six years; Dr. W. J. Denno, medical director of the Standard Oil Company, no cases in seven years; Dr. J. S. Millard, medical director of the Goodyear Tire and Rubber Company, no cases in four years, in 40,000 wounds a year; Dr. E. H. Ingram, medical director of Cramp and Sons, no cases in fourteen years, in 39,000 wounds; Dr. W. B. Fisk, International Harvester Company, one case in twenty-five years, in 36,000 wounds yearly, and Dr. J. Mead, chief surgeon of Ford Motor Company, no cases in last four years, "counting every minor injury, well over a million wounds."

*Summary of Incidence.*—The only conclusion that we can draw from the study of this long series of reports, as well as from the analysis already noted in Table 1, is that tetanus is rare in the ordinary type of industrial wounds. How much of an effort we should make toward prevention will be discussed in the next section.

## ROUTINE PROPHYLACTIC INJECTIONS

Of the 584 surgeons replying to the questionnaires, eighty-three (14 per cent.) employ prophylactic injections of antitetanus serum as a routine in practically all industrial wounds. These men must have been driven to this procedure as the result of bitter experience and it has been interesting to try to determine whether there have been any common factors in forming this group. Why should 14 per cent. of our number adhere to a plan that does not commend itself to the remaining 86 per cent.? The number using routine prophylaxis is large enough to demand consideration; it cannot all be a matter of personal equation. Study of this matter according to the class of industrial work represented does not seem to give any indication that it is special industries that form this group. Only one is connected with a manufacturing plant (hardware, tools and house fixtures). Five are engaged in coal mine work. Among railroad chief surgeons the proportion is greater, nine out of the fifty-seven reporting. There does not seem to be any definite type of industry that induces the surgeons connected therewith to adopt routine prophylaxis. It seems, indeed, that the larger proportion of surgeons advocating routine prophylaxis are those whose work is more especially on the surgical staffs of hospitals than as strictly industrial or plant surgeons. That is, the general surgeon seems more often found in this group than the men following industrial or accident work more as a specialty.

No definite factors connected with industry, therefore, can be found to influence conviction in favor of routine prophylaxis. The study of the data from a geographical point of view is more productive. Of the nine railroad chief surgeons using this plan seven are connected with

roads in the southern states, viz., three in Texas, and one each in Tennessee, North Carolina, Virginia and Mississippi, as against one in New York and one in Illinois. A study of the entire group indicates the same thing. Of the total of eighty-three men in favor of routine prophylaxis in all wounds, only twenty live north of the Mason and Dixon line, a more striking fact when one considers the relative mass of population. The number of surgeons who have for some reason been driven into the group of eighty-three is, for instance, in North Carolina and Texas each ten; in South Carolina, Missouri and Tennessee, each eight. Alabama has seven. Against these states are Massachusetts, one; Michigan, one; Connecticut, one; New Jersey, one, and so on. An explanation for this will be indicated later.

This study embraced (1) the work in which the men in favor of routine prophylaxis are engaged, (2) the incidence of tetanus according to occupation (Table 1) and (3) the reports of industrial surgeons as quoted, and there does not seem to be any one industry or group of industries in which tetanus is more common and in which prophylactic injections are especially indicated.

As to whether the nature of the wound gives any information as to which should be treated with routine prophylaxis one may study Table 2. This gives the nature of the primary wound in nearly 4,000 cases, and indicates sufficiently for a working basis the relative frequency in which various classes of wounds are followed by tetanus.

Punctured wounds make up about one fourth of all cases. Of these the classical punctured nail wound makes up only about half, the remainder being caused by splinters of wood, hypodermic needles, knives, vegetable stubble and thorns, about in the order named. Study of the individual reports does not seem to indicate that in industry even the punctured nail wound of the foot is followed with sufficient frequency by tetanus to warrant routine prophylaxis, especially if prompt and efficient treatment is given to the wound itself. Many surgeons report series of several hundred consecutive cases of punctured nail wounds without tetanus.[1] Several of my correspondents state that they do not fear punctured wounds in ordinary industry, that when promptly and adequately treated they are not followed by tetanus, and that they consider routine prophylaxis decidedly unnecessary.

In the punctured wound I myself do not believe that the tetanus bacillus is often, if ever (except possibly in barnyards and stables), on the puncturing body itself, not even on the classical rusty nail. I think the organisms are on the skin, stocking or shoe, and are driven in at the time of the puncture, or get in later from want of proper surgical dress-

1. Clark: Boston M. & S. J. **176**:541, 1916. Plummer of the Rock Island Railroad: Personal communication to the author.

ing. So that if a punctured wound gets prompt and efficient treatment I do not think routine prophylaxis is necessary unless contamination is present from street dirt, soil from the garden or farm, or from the stable or barnyard.

Unfortunately, perhaps, a close study of this subject does not seem to indicate the practicability of any more systematic plan in the other classes of wounds. Lacerations, burns, compound fractures, crushes and so on in ordinary industry do not show any circumstances that would indicate the possibility of picking out those needing prophylaxis unless, as in the case of punctures, soil, street, stable or barnyard pollution is possible. The child who slips in the garden and barks his shin can as easily get tetanus as if he had received a severe crushing injury. If soil, street, barnyard and stable dirt are barred, there seems no way of telling which of our patients may develop tetanus.

TABLE 2.—*Summary of Nature of Wounds*

| | |
|---|---|
| Puncture wounds | 1,138 |
| Lacerated wounds | 816 |
| Burns | 128 |
| Open fractures | 206 |
| Gunshot wounds (civil) | 169 |
| Crushes | 183 |
| Contusions | 9 |
| Chronic open sores | 56 |
| Superficial conditions | 81 |
| Teeth | 15 |
| Fourth of July accidents | 275 |
| Obstetric | 71 |
| Postvaccination | 218 |
| Medical conditions | 2 |
| Origin undetermined | 112 |
| Wounds by animals | 48 |
| Postoperative | 364 |
| Total | 3,891 |

I do not feel that this study warrants any general suggestion as to the necessity of routine prophylaxis in industry. It will have to remain a local problem which each man must work out for himself. There will be some situations when the proper procedure is obvious. For instance, Dr. William Senger of Pueblo, Colo., reports personally that his service was marked by about two cases of tetanus a year up to eight years ago. Since this time he has used prophylaxis in all cases and there has been no tetanus.

Reference has already been made to the fact that in the South more surgeons seem inclined to routine prophylaxis than in the North. It has also been noted by several writers that tetanus is more common in the summer than in the winter months. These two conditions decidedly harmonize and are undoubtedly due to greater possibilities of soil infection. This would indicate again that tetanus is not essentially an industrial problem but one of the farm, garden, home, street, barnyard and stable; it also indicates the need for more particular consideration of the

question of prophylaxis in these classes of wounds in warm weather or warm climates.

When the nature of wounds is being considered, certain classes deserve a brief note. Many writers and personal correspondents while they do not use prophylaxis in routine cases advise it in all compound fractures. This would seem a somewhat illogical attitude as the number of cases is small and if cases in which soil infection is distinctly mentioned are excluded compound fractures are not more susceptible to tetanus than other severe injuries are.

One of the most distressing points in connection with tetanus is the possibility of its appearing when least expected. One seldom stops to realize that an old varicose ulcer may give rise any day to a fatal tetanus. More disquieting still is the knowledge that in a large number of cases it follows the most trivial injuries, such as the prick by a sewing needle, a blister from a shoe, the merest scratch from a rose thorn or a hypodermic injection.

The number of cases following vaccination while trivial in proportion to the entire number of vaccinations is still far too large. It has been clearly proved that in such cases the infection is not in the virus but that it is always due to later infection of the open sore. It will be rarer if scrupulous care is given the vaccination wound until it heals. Indeed it is probable that in a considerable number of all cases the infection is incurred not at the time of the immediate accident but later. Especially is this true of burns. One case is reported following a lightning burn. After burns by electricity or molten iron, it cannot be doubted that the wound is for a time perfectly sterilized and that contamination creeps in later.

The study of the wounding bodies in injuries followed by tetanus does not give much new information. It is important to note that farm machinery together with farm tools cause nearly as many cases of tetanus as all other classes of machinery.

It is also of great interest to note that after nails, splinters and machinery, the next most frequent wounding body is the hypodermic needle. Many of the cases are in addicts. A considerable number have been reported in India after subcutaneous injections of quinin. It has been considered that in some way the quinin creates a favorable nidus for the development of tetanus spores. In the light of facts mentioned in the foregoing it would seem more probable that these cases are indirect soil contaminations in a warm agricultural country. The most interesting case of all is the so-called "Mulkowa accident." A British medical officer was one day injecting a large number of natives with plague serum. Nineteen developed tetanus. Inquiry showed that this was most probably owing to the fact that the native assistant dropped

TABLE 3.—*Classification According to Wounding Bodies*

| | | |
|---|---|---|
| Puncturing metal bodies | | 697 |
| Nails | 562 | |
| Needles | 4 | |
| Hypodermic needles | 50 | |
| Umbrella needles | 3 | |
| Pitchforks | 14 | |
| Wires | 6 | |
| Knives | 25 | |
| Scissors | 2 | |
| Iron rakes | 6 | |
| Icepicks | 4 | |
| Manure forks | 3 | |
| Pickaxes | 9 | |
| Puncturing nonmetal bodies | | 445 |
| Slivers and splinters | 416 | |
| Vegetable substances (thorn stubbles) | 23 | |
| Bones | 6 | |
| Lacerating and contusing bodies | | 206 |
| Machinery | 85 | |
| Farm machinery | 25 | |
| Saw | 16 | |
| Car wheels | 4 | |
| Glass | 43 | |
| Axe | 12 | |
| Hammer | 5 | |
| Bricks and stones | 7 | |
| Sticks | 4 | |
| Scythe | 2 | |
| Hoe | 3 | |
| Miscellaneous wounding bodies * | | 25 |
| Total | | 1,373 |

* Two each fire poker, sheet iron, horse whip; one each tamping bar, butcher steel, tin can, meat hook, steel cable, spade, chain, iron pipe, binding twine, baseball spike, lawn mower blade, aeroplane propeller, paper file, clam shell, spout of oil can, sled runner, anchor chain, dog collar and hen trough.

TABLE 4.—*Cases Developing After Operation*

| | | |
|---|---|---|
| Unspecified | | 16 |
| Abdominal | | 108 |
| Appendicitis | 37 | |
| Gastro-enterostomy | 1 | |
| Splenectomy | 1 | |
| Cholecystectomy | 3 | |
| Gallbladder | 10 | |
| Acute pancreatitis | 1 | |
| Tuberculous peritonitis | 1 | |
| Inguinal hernia | 38 | |
| Unspecified | 11 | |
| Genito-urinary | | 13 |
| Varicocele | 5 | |
| Hydrocele | 3 | |
| Orchidectomy | 2 | |
| Prostatectomy | 2 | |
| Circumcision | 1 | |
| Gynecologic | | 158 |
| Hysterectomies, abdominal | 29 | |
| Hysterectomies, vaginal | 5 | |
| Myomectomy | 2 | |
| Cesarean | 3 | |
| Radium | 1 | |
| Oophorectomy | 78 | |
| Repair of lacerations | 20 | |
| Alexander | 4 | |
| Ventral fixation | 3 | |
| On appendages | 6 | |
| Curettage | 7 | |
| Rectal | | 31 |
| Hemorrhoids | 23 | |
| Others | 8 | |
| Breast operations | | 17 |
| Amputation of leg | | 9 |
| Sequestrotomy | | 5 |
| Varicose veins of leg | | 2 |
| Miscellaneous * | | 11 |
| Total | | 365 |

* Nasal septum, astragalectomy, nerve suture, ingrown toe nail, eye, tuberculous glands of neck, sebaceous cyst, thyroidectomy, "on leg," kidney (two).

the bottle containing the serum on the ground and it was used without cleaning. One other case developed in another part of India at the same time.

Another most distressing condition in connection with tetanus is its occasional appearance following operation. This accident is still occurring and here again it often follows in entirely unsuspected cases. Table 4 classifies all cases reported in British and American literature for the last twenty years and many personally communicated cases. The question of catgut infection need not be taken up again. In the abdominal group nearly 40 per cent. of the operations are for hernia. In the genito-urinary group nearly all are "clean cases." In the gynecologic group over half do not involve opening either the alimentary or the genital tract.

## CONCLUSIONS AS TO ROUTINE PROPHYLAXIS

I freely confess that this work was undertaken with the idea of compiling evidence that would warrant urging routine prophylactic injections in all industrial wounds. As indicated several times in the foregoing, this does not prove to be justifiable. There are, of course, certain special conditions, as indicated in the South or in Dr. Senger's experience in Pueblo, in which this routine is essential. When, however, many men have gone through a generation of practice in a locality or in an industry and seen thousands of wounds with no tetanus it is idle to tell them that they should begin giving routine prophylaxis to all. It is no more feasible than to give it before every vaccination or operation.

The disease is one of soil contamination and all wounds received in stables and barnyards, all contaminated or possibly so with soil or street dirt, Fourth of July wounds, and gunshot wounds should have it. In these cases a physician who failed to give it might not escape a malpractice verdict.

In ordinary industries we will have to accept the possibility of tetanus arising in the most unexpected cases and rely only on the possibility of improved curative treatment as indicated in the next section.

## BEST METHOD OF TREATING TETANUS

Ever since antitetanus serum was introduced, about 1896, there has been considerable doubt in the minds of many medical men as to whether it has any real curative value after symptoms are established. The war proved beyond question the prophylactic value though additional proof was hardly necessary. The war did not, however, bring forth much conviction as to the value of antitetanus serum as a curative agent. The British seem to have made the most systematic study of

treatment. Sir David Bruce, who was head of the British War Office Committee for the Study of Tetanus, said in an address to the American Red Cross conference in Paris in October, 1918, "There does not seem to be any statistical evidence that serum given therapeutically has marked effect on the rate of mortality." Bruce [2] again repeated this opinion in 1921. Colonel Cummins, who was in charge of the study of tetanus in the British hospitals in France, said at the Fifth International Congress of Surgery, July, 1920, while speaking of a study of case reports in the British Expeditionary Force hospitals, "We were unable to find in these records any clear indication as to the value of antitoxin treatment in tetanus." Captain Golla [3] of the British war office committee said (Lancet, Dec. 29, 1917), "It is a matter of great difficulty in the absence of controls to ascertain whether in addition to the two before mentioned agencies (prophylaxis and improved wound treatment) the specific treatment with serum plays any part in the reduction of tetanus mortality." Bruce during the war said that it was often difficult to establish active treatment by serum as so many of the medical officers actually in charge of the cases considered antitetanus serum useless. A considerable number of American surgeons in correspondence during the last few months have expressed equally doubtful views.

I hope to show that this pessimism is largely due to two factors, first, that antitetanus serum given intraspinally is harmful and diminishes the chances of recovery; second, that when antitetanus serum is given by less harmful methods it is usually given in doses so small that we should not expect it to do any good any way. To develop these two points in detail the opinion first of all expressed is that the intraspinal use of antitetanus serum should be abolished completely and at once.

### USELESSNESS AND DANGER OF ANTITETANUS SERUM INTRASPINALLY

First, it seems universally agreed by the laboratory workers that once tetanus toxin unites with the cells of the central nervous system the antitoxin has no power to break up that union and it would do no good to bathe the cells in antitoxin even if we could do so. Second, there is no evidence that the antitoxic serum injected into the spinal canal gets into the tissues of the cord and brain whether it would do any good or not. Third, it is established that during the disease the toxin is not present in the cerebrospinal fluid so that no toxin is neutralized by spinal injections.

The argument for the spinal canal is that of a geographer based on propinquity, not that of a physician based on physiology. For more

2. Bruce: J. Hygiene **19**:1 (Jan.) 1920-1921.

3. Golla, F.: Tetanus Antitoxin in Experimental Tetanus, Lancet **1**:686 (May 5) 1917.

detailed information as to the uselessness of intraspinal injections theoretically one may refer to the numerous writings of H. E. Robertson of the University of Minnesota. However, as Dr. George P. Muller has said in this connection, no amount of theoretical arguments weigh against well proved clinical tests. In a disease like tetanus the sole clinical test is the effect on mortality.

Tetanus fortunately is so uncommon in the experience of any individual that we can in this disease gain information only from collective studies. The British army surgeons have given us some most important data. Table 5 graphically shows the experience in the British hospitals in France. This form of table was originally used by Colonel Leishman. Its great advantage is that it shows at a glance the result obtained by any method alone or all the methods in any combination. *T* stands for cases treated intrathecally (intraspinally), *V* for intravenous, *S* for subcutaneous, and *M* for intramuscular. The combinations of the capital letters in the first column indicate cases treated by the corresponding combinations of methods of injection. The striking point for our present purpose is that the patients treated intrathecally (*T*), alone or in combination, give a mortality higher by four points in sixty-eight, or about 6 per cent. Not a big increase perhaps, but if the intrathecal method has the advantages heralded for it one would expect a mortality rate at least a little bit lower. Table 6, on exactly the same plan, shows the results for cases in the British home hospitals. Here the patients treated by *T*, alone or in combination, had a mortality higher than those treated without *T* by more than 3:2.

Cases that developed in the British home hospitals were those of decidedly longer incubation period and hence milder and of lower mortality. The cases developing in the British Expeditionary Force hospitals were of shorter incubation, more acute and of higher mortality. It is to be noted that the intrathecal method gives higher death rates in both types of cases. It should be remembered that these cases were not selected but were all the cases on which the committees or reviewing officers could get data. They represent practically a consecutive series. The total number, 1,918, is large enough to eliminate accidental errors. It is most significant that Sir David Bruce, who at the beginning of the war was an ardent advocate for the intrathecal method, said at the end of the war that the experience showed no advantage in it.

Table 7 is the result of an exhaustive review of the literature. In 1905 Anders and Morgan [6] and in 1906 Jacobson and Pearce [7] published articles based on all the literature from the introduction of antitetanus serum to that date, and they also added many personally

6. Anders, J. M., and Morgan, A. C.: J. A. M. A. **45**:314 (July 29) 1905.
7. Jacobson and Pearce: Ann. Surg. **44**:331, 1906.

reported cases. A complete review of the English and American literature from these papers to the present time (including some 600 new articles) is embodied in Table 7. It will be admitted at once that this table shows a rate in favor of intrathecal treatment. However, it was early apparent to me that the general literature is no place to go to find the representative results of various methods of treating tetanus. The

TABLE 5.—*British Army Patients in Hospitals in France (Cummins and Gibson[4])*

| | Cases | Recoveries | Mortality, per Cent. |
|---|---|---|---|
| T | 50 | 7 | 86 |
| V | 11 | 0 | 100 |
| S | 50 | 17 | 66 |
| M | 64 | 28 | 56 |
| T V | 23 | 5 | 78 |
| T S | 65 | 22 | 66 |
| T M | 115 | 39 | 66 |
| V S | 9 | 2 | 77 |
| V M | 6 | 3 | 50 |
| S M | 28 | 10 | 64 |
| T V S | 28 | 7 | 75 |
| T V M | 25 | 4 | 84 |
| T S M | 45 | 20 | 55 |
| V S M | 1 | 0 | 100 |
| T V S M | 9 | 2 | 77 |
| Total | 529 | 166 | |
| Patients (360) treated intrathecally alone or in combination | | | 68.2 |
| Patients (164) not treated intrathecally | | | 64.5 |

TABLE 6.—*British Army Patients in Home Hospitals (Bruce[5])*

| | Cases | Recoveries | Mortality, per Cent. |
|---|---|---|---|
| T | 43 | 19 | 56 |
| V | 33 | 16 | 51.5 |
| S | 248 | 129 | 47.9 |
| M | 133 | 119 | 10.0 |
| T V | 24 | 11 | 54.1 |
| T S | 163 | 88 | 46.0 |
| T M | 250 | 177 | 29.2 |
| V S | 49 | 23 | 53.0 |
| V M | 28 | 18 | 35.7 |
| S M | 85 | 76 | 10.0 |
| T V S | 45 | 31 | 31.1 |
| T V M | 47 | 30 | 36.1 |
| T S M | 205 | 161 | 21.4 |
| V S M | 9 | 3 | 66.6 |
| T V S M | 27 | 22 | 18.5 |
| Total | 1,389 | 923 | |
| Patients (804) treated intrathecally alone or in combination | | | 32.9 |
| Patients (585) not treated intrathecally | | | 20.8 |

literature of this disease is paved with such titles as "A Case of Tetanus Treated by ——— with Recovery." Even the authors own failures by the same or other methods are rarely mentioned. The intrathecal method is so dramatic that an author is much more liable to become enthusiastic and put an intrathecal case on record when he has watched the patient recover than if he had watched the same patient recover with

4. Cummins, S. L., and Gibson, H. G.: Lancet **1**:325 (March 1) 1919.
5. Bruce, D.: J. Hygiene **19**:1 (July) 1920.

sedatives only. No one thinks of reporting his group of intrathecal cases in the literature if the patients all died. Table 7 therefore is of general interest only; it refers largely to selected successes and does not give true mortality rates. It is presented only to give the opposition one argument.

Table 8 is on a sounder basis. It is made up of cases reported to me on special blanks. All are civil cases in America. All are within the last ten years; most are within the last four years. Practically none had prophylactic treatment. Selection is in no way a factor. More than half of these cases are the complete experience of large hospitals. The remainder are the complete experience of surgeons who have been kind enough to contribute their actual results.

TABLE 7.—*Cases Compiled from the Literature*

| | Cases | Recoveries | Mortality, per Cent. |
|---|---|---|---|
| T | 32 | 19 | 40 |
| V | 18 | 8 | 55 |
| S | 451 | 217 | 52 |
| M | 32 | 16 | 50 |
| T V | 20 | 5 | 75 |
| T S | 65 | 31 | 52 |
| T M | 8 | 4 | 50 |
| V S | 26 | 14 | 46 |
| V M | 10 | 6 | 40 |
| S M | 11 | 11 | 0 |
| T V S | 46 | 34 | 26 |
| T V M | 27 | 18 | 33 |
| T S M | 12 | 11 | 8 |
| V S M | 3 | 2 | 33 |
| T V S M | 15 | 15 | 0 |
| Intraneural antitetanus serum | 9 | 4 | 55 |
| Intracranial antitetanus serum | 21 | 10 | 52 |
| Magnesium sulphate only | 61 | 25 | 59 |
| Phenol (carbolic acid) only | 38 | 23 | 39 |
| No specific treatment | 458 | 145 | 67 |
| Total | 1,343 | 668 | 50.3 |

| | |
|---|---|
| Patients (225) treated intrathecally alone or in combination | 39.1 |
| Patients (551) not treated intrathecally | 50.2 |
| Total (806) number of patients treated with serum | 41.0 |
| Total (537) number of patients not treated with serum | 64.0 |

Table 8 again shows a higher death rate for patients treated by intrathecal injections by about 6:5. The mortality in Table 8 for patients treated intrathecally is practically the same as that for patients that received no serum. That large doses intrathecally do not influence the mortality can be seen from the fact that of 190 cases reported to me which were treated with 10,000 units intrathecally, in a single dose the mortality was 59.5 per cent., or practically the same as for patients receiving no serum.

Again, and most important, one should realize that the method of comparison used in the accompanying tables is enormously charitable to the intrathecal method. This is because the vast majority of patients treated intrathecally also had from three to a hundred times as much

serum given by other methods. For instance, a patient may have received 1,000 units intrathecally and 200,000 by other methods. If the patient recovers it must be recorded as a recovery in one of the *T* groups. The mortality of all the *T* groups is thus artificially much reduced. On this account it is indeed most impressive that in Tables 6, 7 and 8 the mortality for the combined *T* groups should, even so, always be higher. Studying this point a little more in detail, we may note how the mortality for any method is increased when intrathecal injections are added. For instance in Table 8 the mortality for intravenous injections (*V*)

TABLE 8.—*Cases Personally Communicated on Author's Blanks*

| | Cases | Recoveries | Mortality, per Cent. |
|---|---|---|---|
| T | 20 | 5 | 75 |
| V | 85 | 31 | 63 |
| S | 38 | 23 | 39 |
| M | 87 | 40 | 54 |
| T V | 106 | 30 | 72 |
| T S | 11 | 4 | 63 |
| T M | 36 | 13 | 64 |
| V S | 21 | 15 | 29 |
| V M | 50 | 24 | 52 |
| S M | 4 | 2 | 50 |
| T V S | 47 | 23 | 51 |
| T V M | 131 | 57 | 57 |
| T S M | 5 | 1 | 80 |
| V S M | 11 | 5 | 55 |
| T V S M | 38 | 18 | 53 |
| No specific | 35 | 13 | 63 |
| Total | 745 | 304 | |
| Patients (243) treated intrathecally alone or in combination | | | 61.7 |
| Patients (297) not treated intrathecally | | | 52.2 |

TABLE 9.—*Increase in Deaths When Intrathecal Treatment is Added to Any Other Method of Treatment (Personally Communicated Cases)*

| | Mortality, per Cent. | | Mortality, per Cent. |
|---|---|---|---|
| V | 63 | T V | 71 |
| S | 39 | T S | 63 |
| M | 54 | T M | 63 |
| V S | 28 | T V S | 51 |
| V M | 52 | T V M | 56 |
| S M | 50 | T S M | 80 |

is 63. When intrathecal injections are added (*TV*) the mortality is 72. That can only mean that the addition of intrathecal injections has killed that many more. This is shown for the entire series of cases reported to me in Table 9. Studies of the British figures show the same thing though not so uniformly.

It is hard to see how the study of Tables 8 and 9 and the British experience can fail to be considered convincing proof that patients treated by intrathecal injections are handicapped and have a higher mortality. Therefore, the first thing to do to cut down the tetanus mortality is to eliminate intrathecal injections.

VALUE OF IMMEDIATE LARGE DOSES BY VEIN

It seems demonstrated that all we can expect from antitetanus serum is to neutralize the toxin circulating in the blood and lymph. To do this the best method theoretically is by intravenous injection. The reason that intravenous treatment has not heretofore given better results clinically is a simple one. It is because in the great majority of cases the dose given is not big enough to do any good. The principle of large doses by vein is in no way original with the author. It has only seemed to him that it is high time that somebody made a determined effort to establish it firmly. Dean [8] of the British army reported fourteen patients treated by doses of 30,000 (U.S.A.) units intravenously. These patients all recovered except one that died under chloroform used while the antitetanus serum was being injected. There was in Major Dean's opinion nothing to show that this death was due to anaphylaxis. In four of the cases no other antitetanus serum was given. In the remaining ten additional doses were given subcutaneously or intramuscularly. Major Dean showed that the patients that later received additional doses (by *S* or *M*) did not do better than those receiving one dose only of 30,000 units by *V*. Dean's patients were all British soldiers and presumably all had prophylactic doses. These patients were in home hospitals. Even with prophylaxis, patients treated by other methods in the British home hospitals never gave a percentage of recoveries like this. Major Dean says that the first effect of large doses by vein is to reduce the pulse rate. Reduction of muscle spasm or convulsion comes later.

An equally convincing article is by Freedlander,[9] who reports four cases in which treatment was given by, respectively, 213,000, 755,000, 189,000 and 188,0000 units, practically by vein only. All the patients recovered. Freedlander has reported to me sixteen other cases from the Cleveland City Hospital, making twenty cases in all, with a mortality of 40 per cent. If the patients dying within twenty-four hours were eliminated, Freedlander's death rate would be 20 per cent. Of course it is not fair to make this elimination, as it is not done elsewhere in this article. Its mention, however, strongly indicates the possibilities. According to our review of Freedlander's reports, the largest number of units given one patient was 755,000. This is the largest number of units in an individual case that we know of. The patient was a boy 8 years old; he recovered. Of Freedlander's patients eight others received over 200,000 units and three received over 600,000 units. None of them showed anaphylactic phenomena. Freedlander's patients were civil ones and did not have prophylactic treatment.

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8. Dean, H. R.: Lancet **1**:673 (May 5) 1917.
9. Freedlander, S. O.: Am. J. M. Sc. **161**:819 (June) 1921.

Here again the crucial test of the argument for large doses intravenously is the effect on the death rate. Table 10 would seem to indicate this conclusively. Thirty-eight representative hospitals reporting five or more cases and using intrathecal injections and small doses by vein report an average mortality of 61 per cent. Freedlander from his hospital, always using large doses by vein, reports a mortality of only 40 per cent. In ninety cases compiled from the literature or reports made directly to me, when 30,000 or more units was used the first day of treatment the mortality was 32 per cent. One can hardly see how the case for large doses by vein, especially as against intrathecal injections, could be made stronger.

It should be noted most particularly that the large dose by vein must be given immediately. Every hour may count. Table 10 shows that

TABLE 10.—*Comparison of Large Doses by Vein* *

| | Mortality, per Cent. |
|---|---|
| 1. 38 hospitals using T or small doses V report 515 cases with | 61 |
| 2. City Hospital, Cleveland (Freedlander), using average initial dose of 78,000 units (largest 100,000) V, reports only 20 cases | 40 |
| 3. 90 compiled cases in which treatment was given by 30,000 units or more V on first day of treatment | 32 |
| 4. 73 compiled cases in which treatment was given by 30,000 units V during the first three days | 55 |

* Hospitals in the foregoing list recording five or more cases: University, Philadelphia; Roosevelt, New York; Massachusetts General, Boston; Episcopal, Philadelphia; Moses Taylor, Scranton, Pa.; Bond's, St. Louis; Buffalo General; Johnstown, Pa.; Erlanger, Chattanooga, Tenn.; Wilkes-Barre General; Pennsylvania State, Ashland, Pa.; Geissinger, Danville, Pa.; Boston City; Lutheran, Fort Wayne, Ind.; St. John's, Springfield, Ill.; Hartford Hospital, Hartford, Conn.; Hamilton General, Canada; Jefferson, Philadelphia; Limon, Costa Rica; State, Nanticoke, Pa.; St. Luke's, Bethlehem, Pa.; University, Augusta, Ga.; Cook County, Chicago; Cooper Hospital, Camden, N. J.; Pennsylvania Hospital, Philadelphia; Presbyterian, New York; Buffalo City; Tourney Hospital, Sumter, S. C.; City, Ithaca, N. Y.; Toronto General, Canada; Touro, New Orleans; New York; Lancaster General, Lancaster, Pa.; Bellevue, New York; State, Scranton, Pa.; Harris, Fort Worth, Texas; Mayo Clinic; Philadelphia General.

if the dose of 30,000 units or more by vein is not completed till the end of seventy-two hours the mortality rate rises rapidly though even so it does not reach the hospital average. McConkey [10] of the Lister Institute, London, indicates the need for immediate adequate dosage when he says that the only way he expects to see progress in the treatment of tetanus is by a closer study of prodromal symptoms.

Of course the proper surgical treatment of the wound, proper nursing in a quiet, dark room, proper sedatives and the like are as important as in any other plan of treatment. I am convinced from the literature that chlorbutanol, as advocated by Hutchins, is the best sedative. Morphin, as H. E. Robertson states, is "probably the worst." This is because morphin blocks all channels of elimination.

10. McConkey: Personal communication to the author.

## CONCLUSIONS

1. Intraspinal injections are harmful, increase mortality, and should be abolished.

2. Antitetanus serum is not useless. Given by vein in doses of from 30,000 to 50,000 units or more, according to the severity of symptoms and the time since onset it will divide the present average mortality rate by two or three, or more. The efficiency of this dose and route will depend directly on the promptness with which it is given.

3. If the initial dose has to be repeated it should be of approximately the same size and by vein only. In the last days of convalescence intramuscular injections are allowable.

4. The best sedative is chlorbutanol, given in a dose of thirty grains (1.95 gm.) dissolved in hot whisky by mouth, or 75 grains (4.87 gm.) in hot olive oil by rectum, repeated sufficiently often to keep the patient relaxed and drowsy till the danger is passed.

NOTE.—In order to obtain more convincing proof of the value of the method of treatment suggested here it is necessary to compile a large number of statistics from various sources. On this account I should appreciate it if any physician who uses this method would report the details and results to me at 912 Clay Avenue, Scranton, Pa.

# THE TREATMENT OF EXPERIMENTAL PERITONITIS BY MERCUROCHROME-220 SOLUBLE

## ALONE OR IN CONJUNCTION WITH HYPERTONIC DEXTROSE SOLUTIONS *

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With the demonstration of the etiologic rôle of bacteria in disease, the search for specific remedies could be more intelligently directed; nevertheless, it is a source of both surprise and discouragement to realize how few of these have been placed at our disposal since Pasteur's momentous discoveries. Particular significance is attached to the suggestion of Piper [1] that in mercurochrome-220 soluble, intravenously administered, we may possess a specific against various blood stream infections. Young,[2] under whose direction this drug was first introduced into therapeutics, immediately acted on Piper's suggestion, and the successes obtained by this author and his co-workers appeared so brilliant that they were, indeed, "tempted to soar into the realms of fancy" and conclude that in mercurochrome-220 soluble they saw the realization of the age old dream of "therapia magna sterilisans," through which could be healed all the ills of the flesh. Unfortunately, however, not all clinical reports on the use of mercurochrome-220 soluble intravenously have been so charged with enthusiasm as the earlier papers by the Baltimore investigators.

In a recent publication, Young attempts to analyze the results obtained by observers in widely scattered localities: some of these are distinctly discouraging, not only because they appear to indicate that the drug failed to exert a beneficial action, but also because in a few of them the death of the patient was attributed to the effects of the drug. Since the appearance of Young's paper, a contribution to the subject has been made by Brill and Myers [3] which would lead one to infer that the drug

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* From the experimental surgical and pharmacology departments of the Medical College of Virginia.

1. Piper, E. B.: The Treatment of Puerperal Sepsis by the Use of Mercurochrome Intravenously, Am. J. Obst. & Gynec. **4**:532 (Nov.) 1922.

2. Young, H. H., and Hill, Justina: The Treatment of Septicemia and Local Infections by Intravenous Injections of Mercurochrome-220 and of Gentian Violet, J. A. M. A. **82**:669 (March 1) 1922.

3. Brill, I. C., and Myers, H. B.: Mercurochrome-220 Soluble and Gentian Violet: Bactericidal Efficiency by Intravenous Route, J. A. M. A. **84**:879 (March 21) 1925.

is entirely without value in the treatment of bacteremias and of localized infections of the genital tract when administered intravenously.

An unbiased reader, taking into consideration all the various reports, favorable and adverse, can scarcely escape the conclusion that striking results have been associated with the intravenous use of mercurochrome-220 soluble, and that these results have followed the administration of the drug so closely that it is fair to assume that this administration was the cause, of some at least. On the other hand, it seems just as positively shown that the early enthusiasm expressed by some writers was scarcely justified; mercurochrome-220 soluble administered intravenously not only fails to effect a cure in a considerable proportion of the cases but it, not infrequently, causes a severe reaction, rather prolonged toxic manifestations of a mild character, or even death.

In view of the diverse conclusions that have been drawn from clinical observations, it seems indeed remarkable that so little animal experimentation has been carried out in the attempt to arrive at definite conclusions regarding the therapeutic efficiency of intravenously administered mercurochrome-220 soluble. It is true that Piper reported experiments on rabbits,[1] but, although he and his associates used some thirty animals, he seems to rely solely on one pair; one of these became critically ill after receiving an intravenous injection of an emulsion of streptococci, to recover in a spectacular manner under mercurochrome therapy, while the untreated control succumbed in thirty hours. As impressive as may be the recovery of a rabbit, "comatose and with an eye full of pus" following the injection of mercurochrome-220 soluble, a single instance of such an occurrence cannot carry much conviction. Rabbits have become critically ill from the injection of virulent microorganisms and have then recovered, though denied the benefit of mercurochrome-220 soluble treatment; of two untreated animals, one may succumb from a dose of streptococci while a second may survive. Experiments such as Piper reported are of value when their number is comparatively large; only in this way can the element of chance be discounted. Because of the great diversity of opinion expressed by clinicians and the absence of satisfactory experimental evidence, it was deemed advisable to carry out experiments on the use of intravenously administered mercurochrome-220 soluble in the treatment of experimental peritonitis.

In this investigation, dogs have been used exclusively. Peritonitis was produced as follows: Under morphin-ether anesthesia, an incision about 5 cm. long was made through the wall of the large intestine, low in the pelvis, and the abdominal wound was closed by through-and-through

silk sutures. The death of the animal usually occurred within twenty-four hours; at necropsy, there was present evidences of a marked inflammatory reaction of the peritoneum, the membrane being intensely congested and the peritoneal cavity containing a varying amount of hemorrhagic exudate. If this exudate was removed and a dose of from 3 to 4 c.c. per kilogram of body weight injected intraperitoneally into a second dog, it usually caused the death of this animal in from eighteen hours to three days. At necropsy, this second dog generally presented the same picture as that described for the first one; in rare instances, the exudate was reddish grey or was frankly purulent in nature. The exudate from the second animal was found to be of greater toxicity than that of the first; by continued passage through dogs, its virulence could be greatly increased; it having been possible to obtain a specimen that would prove fatal to the great majority of animals in a dose of 0.1 c.c. per kilogram of body weight after intraperitoneal injection.

The exudate as first obtained contains a varied bacterial flora. Dr. G. F. Reddish has demonstrated the presence of *Bacillus coli, Bacillus welchii,* streptococci and enterococci; more recent bacteriologic studies carried out by Dr. F. W. Shaw show that *Bacillus neapolitanus* is found in almost pure culture after repeated passage of the fluid through dogs. Important as the bacteria may be in bringing about death following the injection of the exudate, nonliving toxins, also, must be reckoned with. That this is the case has been demonstrated by injection of the filtrate obtained after subjecting the exudate to a boiling temperature for fifteen minutes: this filtrate, in sufficiently large dose, will cause death with all the symptoms following the injection of the unchanged exudate.

In the mercurochrome-220 soluble experiments, the ideal would be to inject the animals with an amount of the exudate that would cause the death of all the untreated controls. This has been found impracticable; dogs with unusually high resistance would necessitate the injection of an amount of the exudate that would invariably prove fatal, regardless of the method of treatment employed. It has been necessary, therefore, to attempt to use a dose that will produce a high mortality in the controls, and to note the effects of mercurochrome-220 soluble after the injection of such a dose of the exudate in other animals. Difficulty is encountered here; even though kept in an icebox, the exudate gradually decreases in potency; the dose employed may be either too small to kill any of the controls, or, on the other hand, so large as to cause the death of all the animals, controls and treated alike.

In a few of the earlier experiments, the mercurochrome-220 soluble was used in the form of a 2 per cent. solution in distilled water; later, a 1 per cent. solution was employed. The mercurochrome-220 soluble

solution was injected either into the marginal ear vein or into the external jugular; the lapse of time beween the intraperitoneal injection of the exudate and the intravenous injection of the mercurochrome-220 soluble varying from two to eighty-eight minutes. Following the advice of Young, the dose of 5 mg. per kilogram of body weight was administered in all the experiments; from this dose, we never observed effects of an undesirable nature. In practically every instance in which the animal survived more than twenty-four hours, this dose was repeated daily until death or apparent recovery was manifest.

Owing to the difficulties in connection with selection of a suitable dose of the exudate, a large number of dogs have yielded results of no value, the amount of the exudate injected being either too small or too large. After eliminating these unsatisfactory experiments, forty-six animals remain, twenty-one controls and twenty-five treated dogs, permitting fairly definite inferences. The results of these experiments are given in Table 1.

TABLE 1.—*Effect of Mercurochrome-220 Soluble in Experimental Peritonitis: Mortality of Control and of Treated Dogs*

| Exudate | Dose of Exudate, C.c. × Kg. | Controls | | | Treated | | |
|---|---|---|---|---|---|---|---|
| | | Survived | Died | Mortality, per Cent. | Survived | Died | Mortality, per Cent. |
| A | 0.25 | 1 | 1 | 50 | 2 | 2 | 50 |
| A | 0.30 | 1 | 1 | 50 | 1 | 1 | 50 |
| B | 0.25 | 0 | 1 | 100 | 2 | 0 | 0 |
| AB | 0.50 | 0 | 1 | 100 | 2 | 1 | 33 |
| A | 0.50 | 2 | 7 | 78 | 3 | 5 | 62 |
| B | 0.50 | 2 | 2 | 50 | 3 | 1 | 25 |
| X | 0.50 | 1 | 1 | 50 | 0 | 2 | 100 |
| Totals | | 7 | 14 | 66 | 13 | 12 | 48 |

These results would appear to indicate that the intravenous injection of mercurochrome-220 soluble had a slightly beneficial effect when used in the treatment of experimental peritonitis. Certainly, we have never observed anything "spectacular" about the action of the drug in this condition; indeed, we are not at all convinced that the lower mortality in the mercurochrome series was actually due to this drug. Certainly, if mercurochrome-220 soluble exerts a desirable action in experimental peritonitis, it is slight; as indicated by the results obtained through the use of an exudate not included in Table 1.

This exudate was administered in the dose of 0.45 c.c. per kilogram to four controls, all of which recovered. The dose of 0.5 c.c. per kilogram given to three dogs and followed by mercurochrome therapy was responsible for a fatal outcome in all three instances.

Striking results have been reported by certain of the German investigators to have followed the intravenous injection of hypertonic dextrose

solutions. Of especial interest are the observations that such injections may serve to increase the bactericidal action of drugs used contemporaneously: because of the somewhat discouraging results that had been obtained by the use of mercurochrome alone, it was decided to attempt to ascertain whether the supposed "spectacular" action of this drug could be brought out by its use in conjunction with hypertonic dextrose. The experiments bearing on this point were carried out in the same manner as those previously described; various specimens of exudate were injected intraperitoneally, to be followed by the intravenous injection of mercurochrome in the dose of 5 mg. per kilogram of body weight. In addition, the treated dogs were given 10 c.c. of 25 per cent. solution of dextrose per kilogram of body weight, this injection being made into the jugular vein and immediately preceding or following the mercurochrome injection. The results of these experiments are given in Table 2.

So far as these results permit drawing conclusions, they are distinctly unfavorable to the use of mercurochrome-220 solution and hyper-

TABLE 2.—*Effect of Mercurochrome-220 Soluble and Dextrose in Experimental Peritonitis: Mortality of Control and of Treated Dogs*

| | | Controls | | | Treated | | |
|---|---|---|---|---|---|---|---|
| Exudate | Dose of Exudate, C.c. × Kg. | Survived | Died | Mortality, per Cent. | Survived | Died | Mortality, per Cent. |
| B..... | 0.75 | 5 | 1 | 16.6 | 4 | 2 | 33.3 |
| B..... | 1.50 | 3 | 3 | 50.0 | 0 | 7 | 100.0 |
| C..... | 1.00 | 1 | 1 | 50.0 | 1 | 1 | 50.0 |
| Totals..... | | 9 | 5 | 35.7 | 5 | 10 | 66.6 |

tonic dextrose solutions in the treatment of experimental peritonitis. It is certainly not justifiable to assume that the mercurochrome was responsible for the unfavorable effects; the outcome where this drug was used alone in the treatment of experimental peritonitis speaks against this. Possibly the strongly hypertonic solution of dextrose injected into the blood vessels hastened absorption from the peritoneal cavity, thus overwhelming the extraperitoneal defenses of the body.

The outcome in these experiments in which mercurochrome-220 soluble was used alone in the treatment of experimental peritonitis would scarcely make one infer that the drug possesses "marvelous" curative properties; nor lead even to the conclusion that it constitutes a "really remarkable addition to therapeutics" so far as regards this pathologic condition. In all fairness to mercurochrome-220 soluble, it should be again emphasized that peritonitis presents an instance of *toxemia*, as well as bacteremia; the advocates of mercurochrome therapy have never claimed that the drug would be efficacious against toxins. In clinical peritonitis, when the exudate is removed operatively, it is at least con-

ceivable that the intravenous injection of mercurochrome-220 soluble would prove of more benefit than was the case in the experimental form; although the few cases already on record do not prove this. On the other hand, we should not lose sight of the fact that in a patient suffering from diffuse peritonitis, the condition is often critical and the intravenous injection of a substance with the capabilities of doing harm to the extent that mercurochrome-220 soluble seems to have manifested on at least three occasions may actually contribute to or cause the fatal outcome.

# TWENTY-NINTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

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## CONGENITAL CONDITIONS

*Developmental Anomaly of Patella Frequently Diagnosed as Fracture.*—Adams and Leonard[1] call attention to the fact that the patella is a sesamoid bone and thus subject to congenital anomalies that may be mistaken for fractures. Congenital anomalies of the patella are more common than has generally been supposed. In a series of sixty-three cases of fractured patella during one year, more than 3 per cent were congenital anomalies erroneously diagnosed. The differential diagnosis between a congenital anomaly and fracture can easily be made from a roentgenogram. These deformities are usually bilateral. The adjacent surfaces of the bone are smooth and consist of cortical bone. The congenital anomalies are located in the outer and upper quadrant, which is a rare location for fracture. The absence of a typical clinical history and the symptoms usually associated with fracture should make the examiner suspicious that he is dealing with a congenital anomaly. The recognition of the possibility of this congenital anomaly is important for economic reasons, if for no other.

## TUBERCULOSIS

*Spur Formation in Caries of the Spine.*—Sorrel[2] and Duguet and Clavelin[3] in different articles call attention to three cases of beaklike spur formation at the seat of lumbar caries. Sorrel's patients were aged 20 and 25. In Duguet and Clavelin's case the question was raised of a

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*This report of progress is based on a review of 357 articles selected from 753 titles dealing with orthopedic surgery, appearing in medical literature between July 1, 1925, and December 12, 1925. Only the papers that seem to represent progress have been selected for note and comment.

1. Adams, J. D., and Leonard, R. D.: Surg. Gynec. Obst. **41**:601 (Nov.) 1925.
2. Sorrel, E.: Bull. et mém. Soc. nat. de chir., July 11, 1925, p. 798.
3. Duguet and Clavelin: Bull. et mém. Soc. nat. de chir., July 11, 1925, p. 801.

co-existing hypertrophic arthritis, but Sorrel's patients were too young for this disease to develop.

*Roentgen-Ray Treatment of Bone Tuberculosis.*—Southard [4] has treated twenty-seven cases of bone tuberculosis with the roentgen ray. The cases involved twelve different bones or joints; 81.13 per cent of these seemed to have been "cured" for one or more years before this report. In the treatment no massive doses are needed or used. The author's theory is that there is nothing one desires to kill by the rays. The field is already strewn with dead and dying cells. The indication is to stimulate, nourish and encourage the living cells and tissues to dispose of the dead by elimination or absorption, while at the same time the phagocytes are invited and encouraged to feast on the tubercle bacilli present. The writer does not feel that the number of cases reported is "sufficient to base any final conclusion on, but he offers them merely as pointing to a safe, sane and effective treatment of a most intractable disease which has heretofore been resistant to other methods of treatment and which has crippled and is crippling untold thousands of our race, both adults and children."

[ED. NOTE.—Reports of the beneficial effects of roentgenologic therapy in tuberculosis of the bones and joints continue to appear from time to time. One of the editors has had an experience in a proved tuberculous elbow joint which slowly healed, discharged a sequestrum while undergoing this treatment, excised itself, as it were, and has remained healed with limited but useful motion for ten years. There has been no convincing research as to the effect of roentgen rays on tuberculous bone and joint lesions. Perhaps it should be undertaken. Before any conclusions as to the value of this form of treatment can be drawn we must have laboratory proof of the nature of the joint lesion.]

*The Treatment of Tuberculous Abscess and Sinus.*—Starr's [5] long experience in the treatment of tuberculous abscesses and sinuses and his faculty for keen observation command attention to his conclusions. He believes that the great danger from all forms of treatment, such as aspiration, aspiration with injection of antiseptics, incision and drainage, is the danger of secondary infection, which he considers great. For many years he has followed out a routine in all tuberculous abscesses. The abscess is treated expectantly, being carefully watched while the bone lesion is being treated. In a large proportion of cases the abscess becomes a thickened or calcified mass. Surgical intervention is advised in the cases in which in spite of careful treatment of the original bone lesion the abscess gradually increases in size and ultimately threatens to break down the skin. At this time under most careful asepsis an incision

4. Southard, J. B.: Radiology **5**:34 (July) 1925.
5. Starr, C. L.: M. J. Australia, Aug. 15, 1925, p. 183.

is made through healthy skin and a muscle layer if possible. When the sac is reached, the content is aspirated in such a manner as to prevent soiling the wound. The cavity is then opened widely and the granulations cleaned with gauze. The bleeding is stopped by pressure and the cavity swabbed with iodine. The débris is wiped out with alcohol and the wound sewed up in layers. If the abscess should fill up again, the same procedure is repeated. Since this treatment has been his routine, amyloid disease following bone and joint lesions has been practically unknown and tuberculous meningitis rare. In the treatment of sinuses there are two essential rules of conduct: first, no one should put anything into the sinus for any purpose; second, the sinus should be dressed as infrequently as possible, and when these dressings are necessary the area should be kept clean and the dressing should be done as aseptically as possible.

*Tuberculous Disease of the Spine: Conservative Treatment.*—Gauvain [6] gives a chart showing an analysis of 192 cases of surgical tuberculosis which shows that in over 60 per cent of cases of spinal caries originating in children under 10 years of age the children were infected from bovine sources. Above 10 years the incidence of infection from bovine sources diminishes rapidly. He believes that abscesses from spinal caries, with rare exceptions, should not be widely opened. He does not consider it wise to leave an abscess to absorb, as he believes that it tends to the spread of the disease and destroys the tissue with which it comes in contact and that it increases toxemia and delays cure. Where the abscesses are accessible, he advocates that they should be aspirated as soon as possible after their formation. Occasionally their aspiration may be assisted by the employment of modifying fluids. He thinks the method of Calvé the best. Fusion operations on children he thinks have little value in shortening the period of treatment. They are by no means devoid of danger unless prolonged and careful after-treatment is instituted. They will not prevent the onset of deformity and will not even insure against relapse. He believes it is possible to correct by special apparatus some of the deformity of the spine in children, but says that the day of forcible correction is past.

[Ed. Note.—From some inconclusive but suggestive studies in centers of population in America where the milk supply is well supervised, it seems probable that bovine tuberculosis is playing a less important part in our bone and joint lesions than we had supposed. One of the editors on a visit to Norway this summer was surprised to find that a well known orthopedic surgeon, after a careful investigation, had become convinced that tuberculosis in cows was a rare disease in Norway and

6. Gauvain, Henry: Brit. M. J. **2**:937 (Nov. 21) 1925.

that the percentage of bovine tuberculosis in children was practically negligible, and yet the surgical tuberculosis of children seemed to be common throughout the country.]

*Tuberculous Disease of the Spine: Operation for Spinal Fixation.*—Girdlestone [7] believes that spinal fixation by operation in children should be employed in certain cases and at certain ages of the disease; namely, when the caries has led to such instability of the column as will through strain cause progressive deformity. This is difficult to check by splintage and may for the same reason lead to a recurrence of the disease. It may also be employed when a disease has been rendered quiescent after several years of recumbency. In adults he says that the operation is most valuable as a part of the conservative treatment. He believes that an angular deformity is to be expected when there has been much loss of bone; that such an angular fusion demands protection during the efforts to produce compensatory curves, rather than an attempt to open the angle, which is theatened by the methods advocated by Sir Henry Gauvain, unharmful as these have seemed to be in Sir Henry's hands. Girdlestone quotes Rollier that "to open and drain a cold abscess is to commit a surgical crime. It is almost a condemnation to death, not at once, but in two or three years." He urges the need of beds for the care of adults with spinal tuberculosis.

*Treatment of Knee Joint Tuberculosis.*—Hibbs and von Lackum [8] have followed up the results of seventy-seven patients with knee joint tuberculosis who came to their hospital during a period of seventeen years. Of the seventy-seven patients, seven are dead. Of the seventy remaining, sixteen have been operatively fused. Of these twelve were cases in which relapse had taken place, and four were cases that did not become quiescent. The disease became quiescent in fifty-three patients, including two who died; fifty-one remain, in ten of whom the diagnosis of tuberculosis was probably incorrect, leaving forty-one. Of these, sixteen have already relapsed, and twelve of them have been fused; two are again quiescent, and four remain active. Add to these the nine that never became inactive and there are still thirteen active cases in the whole series. Twenty-seven cases are inactive, three with bony ankylosis, and therefore safe; two with fibrous ankylosis and unsafe, and twenty-two with varying degrees of motion and deformity, all of which are certainly unsafe. The average of onset was four years. The duration of time before the quiescent period was reached was on the average six and one-half years. The average period at which relapse has occurred in quiescent cases was seventeen years, and seven years

7. Girdlestone, G. R.: Brit. M. J. **2**:940 (Nov. 21) 1925.

8. Hibbs, R. A., and von Lackum, H. L.: J. A. M. A. **85**:1289 (Oct. 24) 1925.

after discharge from the hospital. The relapses to date among the cases that become quiescent are well over one-third of the series. The average period during which these patients have been observed is over fifteen years, the longest twenty-four and the shortest six years. The writers conclude from this study that the methods of diagnosis used in these cases were wholly untrustworthy. Guinea-pig inoculation of aspirated fluid or tissue is the only method that is positive. They say, "We are forced to the conclusion from this series at least that the conservative methods here used have failed to accomplish a cure in all but three cases that were fused by nature. The other cured cases, sixteen, were fused by operation. The elimination of motion by fusing the knee by operation offers the only means we know of curing the disease, and it should be done early in the disease, and may be done as early as the sixth year without any disturbance of the growth of the leg."

[ED. NOTE.—On the basis of the author's figures, we agree that their conclusions are warranted, and yet most of us are unwilling to accept them; nor do we consider ourselves justified in fusing every proved tuberculous knee in children from 6 years old until the growth period stops. Calvé, Rollier, Lo Grasso and other observers who have carried out their principles do not believe that knee joint tuberculosis in children can only be safely cured by bony ankylosis. They can show—indeed, we ourselves have seen—proved cases with arcs of motion far beyond the merely useful range; the patients for several years have been leading the athletic, traumatizing life of active boys without the suggestion of a recurrence. To be compelled to feel that it is conservative surgery to stiffen operatively all proved tuberculous knees in children more than 6 years of age is after all a confession of failure. A stiff knee is an irrevocable handicap. Let us be more sure than the editors are at present that there is no other "way out." The article is a delightfully frank and thorough review and should stimulate much thought and, we hope, attempts to provide opportunity for the wider use of the sunshine and open air method of combating the constitutional disease, and perhaps of safely conserving joint function.]

### POLIOMYELITIS AND OBSTETRIC PARALYSIS

*Poliomyelitis with Polyarthritis.*—Saethre[9] reports a case of poliomyelitis associated with joint affections and with severe spontaneous pains of long duration in the limbs. The right elbow and the right hip joint were swollen, and any attempt at motion painful; the basal joint of the right thumb, both shoulder joints, and the right knee were slightly affected. The pains were of continuous, burning character, and involved the right leg and thigh. The joint affections greatly improved with

9. Saethre, Haakon: Norsk. Mag. f. Lægevidensk., Sept. 1925, p. 966.

salicylates and disappeared from nine to ten weeks after the onset of the disease. Attacks of pain persisted for seven months and often required sedatives. In discussing the unusual symptoms, the author states that the pains might have been caused by a meningeal affection. As to the joint phenomena, he thinks it most probable that they were of toxic, hematogenous origin and were due to the poliomyelitis virus. The possibility of a spinal form of epidemic encephalitis was considered.

*Convalescent Serum in Preparalytic Cases of Poliomyelitis: Results of Intramuscular Administration.*—Shaw, Thelander and Fleischner [10] state that the intramuscular injection of convalescent serum is at least theoretically a means whereby passive immunity may be achieved in a manner similar to that observed in the development of active immunity; namely, through the blood. They report five cases, and although they realize the danger of drawing conclusions from so small a number, they feel justified in concluding that the evidence indicates that preparalytic cases of poliomyelitis, even when seemingly of the fulminating or malignant type, can be cured by intramuscular injections of fresh convalescent serum if it is administered in adequately large doses at a sufficiently early period of the disease.

[ED. NOTE.—It is fair to state that on the basis of animal experiments most research workers who have attempted to evaluate the curative effects of convalescent serum do not feel justified in drawing the conclusions of the authors, nor, on the other hand, are they willing to make the statement that it never exerts any favorable influence.]

*Public Health Aspects of Poliomyelitis.*—Ebright [11] believes that the most efficient agency of control of an epidemic is the public health nurse. Patients are to be isolated in the acute stages in much the same way that typhoid patients are, with similar precautions regarding body discharges. Nose and throat sprays of antiseptic solutions are useless in that they do harm in removing a protective substance in the nasal secretion which neutralizes the virus of the disease. The closing of schools is advised against. A study of the 1925 epidemic in California showed that the number of cases was greatest about the first week in August, and in spite of the return of over 200,000 pupils to school at that time, the number of cases in the succeeding two months steadily diminished.

*Obstetric Paralysis: Report of Eleven Hundred Cases*—Sever [12] gives an interesting analysis of 1,100 cases of obstetric paralysis. Of these, 551 were in boys and 549 in girls. The right arm was affected 670

10. Shaw, E. B.; Thelander, H. E., and Fleischner, E. C.: J. A. M. A. **85:** 1555 (Nov. 14) 1925.

11. Ebright, G. E.: Report of meeting of San Francisco County Med. Soc., 1925, abstr., California & West. Med., November, 1925, p. 1428.

12. Sever, J. W.: J. A. M. A. **85:**1862 (Dec. 12) 1925.

times; the left 430, or about 64 per cent in favor of the right. The upper arm type occurred 829 times, as against 208 of the lower arm type. In the series 584 cases were definitely recorded as long and difficult labors. There were sixty-three cases in which both arms were affected, the whole or lower arm type being the least common. In a large proportion of the 584 cases forceps were used, either high or low, and most of the mothers had ether, which presupposes a relaxed and asphyxiated child. These are ideal conditions for the production of such a paralysis.

Roentgengrams in the early stages have shown nothing except an occasional fracture of the clavicle or humerus. No epiphyseal separations or dislocations appeared. In the later stages the shaft of the humerus is smaller; the epiphyseal head of the humerus is a little smaller than the one on the well side. The glenoid fossa becomes flattened, particularly in the older babies who have a posterior dislocation. At this time also there is a hooking of the acromion more or less marked, depending in its extent on the degree of subluxation. The treatment should be begun immediately and should be carried on faithfully for years. It is better supervised by a trained person than by the parents. Except in the whole arm type, a fairly good functional arm can be expected. Patients when first seen should not have the arms put in a sling or a Velpeau bandage, but should be left entirely free, with the other arm pinned, or should be put on a splint. In old cases in which there has been no treatment, operation must be performed almost invariably. Operations designed to relieve muscle contracture should not be done earlier than the age of 4 or 5 years. Sever does not take as radical a position in regard to operations on the brachial plexus as formerly. He is more willing to advise operation in those of the upper arm type in which one or two muscles seem permanently paralyzed or so much weakened that they cannot function successfully. In the whole arm type when there is no functional improvement at all after three months of muscle reeducation, an operation on the plexus is indicated, but nothing is to be promised. In the upper arm type he often waits until the child is 3 or 4 years of age because at that time muscle contractures can also be divided.

### ARTHRITIS

*Rheumatoid Arthritis: Etiologic and Bacteriologic Aspects.*—Rolleston [13] discusses whether infection is the sole and exclusive factor in rheumatoid arthritis. After consideration of the metabolic findings, such as low sugar tolerance, lowered metabolism, as described by Pemberton, and the endocrine theory of Draper and H. K. Thompson, the writer believes that they can all be explained by an underlying

13. Rolleston, Humphrey: Brit. M. J. **2**:589 (Oct. 3) 1925.

chronic infection, and are not themselves primary causes of arthritis. To substantiate the infective origin, he mentions Hunter, Billings, Willcox, Pemberton and Watson-Williams as authorities believing in aural sepsis. He instances pyorrhea and apical infection, sinuses and tonsils, and believes that intestinal infection depends on achylia gastrica. He mentions Mutch's finding of intestinal staphylococci in Still's disease and streptococci of glycophile character as of interest in relation to a low carbohydrate diet and its results. He thinks that few colon infections not secondary to an aural infection are responsible for arthritis, owing to the antitoxic functions of the liver. Any infection, gonorrheal or other, if located in prostate and seminal vesicles, may cause arthritis. Streptococcic endocervicitis may also produce it. The tuberculous, chronic pulmonary osteo-arthropathy of the French authors has little support in England, but cannot be overlooked as a possibility. That skin infections may cause arthritis, boils, dermatitis and impetigo is questionable. Arthritis associated with psoriasis may be a toxic arthritis only. Infection depends on natural immunity or type susceptibility. Any change in resistance may open the field. Unless all foci are removed, results cannot be expected. Sir Humphrey thinks that various streptococci are most frequently responsible and the staphylococci less often. The most serious objection to the infective theory is the inability of most observers to find organisms in the synovial fluid or to obtain cultures from the synovial membrane. The treatment of the disease consists in removal of the possible focal causes cautiously and perhaps in the use of autogenous vaccine and intestinal antiseptics, endocrine medication when its administration is definite, and of Pemberton's dietetic treatment. The orthopedic aim is to prevent deformity. Heliotherapy and ultraviolet radiation may be helpful. Protein shock therapy from vaccines of any kind, milk, bee's sting, etc., seem to afford only temporary relief.

*Sulphur Metabolism in Rheumatoid Arthritis.*—Cawadias [14] calls attention to the fact that sulphur is eliminated by the kidneys. The urine, therefore, if examined for sulphur is an index of sulphur metabolism. An excess of sulphur is apparently eliminated in this type of arthritis. The normal excretion is from 2.5 to 3 grains (0.16 to 0.19 Gm.) of sulphur ($SO_3$) per day. The arthritic excretion is from 4 to 5 grains (0.26 to 0.32 Gm.) per day, and this cannot be interpreted as simply a sign of protein destruction because the nitrogen does not keep pace. The tissues of the rheumatoid case do not seem to be able to hold sulphur. There is also a relative increase of neutral or imperfectly oxidized sulphur. The normal figures are from 14 to 20 per cent of the total urinary sulphur, while in arthritis Cawadias finds from 25 to 33 per cent, owing, he thinks, to the relative decreased oxidation of the

14. Cawadias, A.: Brit. M. J. 2:602 (Oct. 3) 1925.

organism as in hydrocyanic acid poisoning. The external sulphates are not increased; this suggests that the increase in sulphur excretion is not from cartilage destruction, but from a general cellular metabolism defect: (*a*) syndrome of sulphur demineralization; (*b*) syndrome of sulphur hypo-oxidation. Metabolic trouble may be due to infection, but is preceded by a tendency to hypo-oxidation or toward sulphur demineralization.

*Influence of Focal Infection and the Pathology of Arthritis: Results of Experiments.*—Pemberton and his co-workers [15] proved satisfactorily to themselves that there is no support for the theory of an increased rate of absorption from the intestinal tract in arthritis. He then worked on the theory that the blood inadequately reaches some of the tissues and that this might result from the closed capillary beds and would leave some of the blood constituents unutilized. This hypothesis was tested by interference through the posture with the blood flow in the limbs of arthritic patients and normal persons, and a glucose tolerance test was then conducted. The results showed that in half the cases studied it was possible to induce a lowered sugar tolerance in subjects presenting otherwise a normal sugar tolerance. In view of the close relationship of a lowered sugar tolerance to arthritis and focal infection, this evidence approaches proof that a disturbance of the circulation, probably in the nature of closed capillary areas, constitutes an important part of the pathology of arthritis and the rheumatoid syndrome. If these findings are correlated with the well recognized benefits accruing from measures that improve the circulation, the evidence becomes stronger.

*Operative Treatment of Arthritis Deformans of Hip Joint.*—Whitman [16] observes that the operative treatment of ostearthritis of the hip is practically limited to cases of confirmed disability in which the other joints are free from disease, at least in a disabling sense. The attitude of election of the limb after arthrodesis is one of slight flexion, abduction and outward rotation, but in a large proportion of the cases, because the fixation is incomplete, there is a gradual relapse toward the original attitude of adduction. Minor disadvantages of arthrodesis are that patients require assistance in dressing, while the stiffened joint increases the strain on the lumbar spine, and is thus in some instances a cause of discomfort. It would seem, therefore, that the most practicable, and in most instances the preferable, alternative to destruction of the joint is a reconstruction operation. In place of excision of the head, its cartilage and the underlying softened bone are removed until

15. Pemberton, Ralph; Cajori, F. A., and Crouter, G. Y.: J. A. M. A. **85**: 1793 (Dec. 5) 1925.

16. Whitman, R.: Ann. Surg. **81**:1108 (June) 1925.

fairly resistant tissue is reached, reducing its size to about the diameter of the neck. The trochanter is cut at its base in the line of the angle of the neck and transplanted to the outer side of the shaft. The removal of the trochanter permits the extremity of the neck to be thrust deeply into the acetabulum where the cartilage is often in fairly normal condition. By reducing the size and changing the bearing surface of the femur one may check mutual friction and yet retain motion, and by restoring the leverage of the abductor muscles, the adduction deformity is prevented. The technic of the reconstruction operation has not been changed essentially from the original description. As compared with arthrodesis as an established surgical procedure, the reconstruction operation is still in the tentative stage. It is far more difficult than arthrodesis, more especially because the subjects are usually elderly and often obese. Yet in twenty-five cases there has been only one death, from embolism, an accident that has no relation to the character of the operation. Other than this, there has been no serious complication. A direct comparison of the results of the two operations is difficult, because in one the primary object is to relieve pain by destroying the joint; deformity and incidental disability are secondary considerations. In the other the purpose is to restore a useful degree of joint function. It is obvious that bony fixation must be the surest remedy for pain caused by friction, for whenever the free range of motion in a joint is restrained from any cause, sudden strain on the restricting tissues causes discomfort. To this, however, the patient accommodates himself and would rarely exchange even limited motion for fixation, for it is this mobility which checks deformity, which is so common after arthrodesis, and which enables the patient to sit with comfort and to walk with a fairly symmetrical gait. From a practical standpoint it may be stated that the majority of the patients have been women, all of whom, except those under immediate treatment, have been able to resume their household duties; not one of the entire number was disappointed with the result, either immediate or remote. Whitman thinks, therefore, that the reconstruction operation may be considered as the preferable alternative to arthrodesis for all cases of a favorable type.

### RICKETS AND BONE DISEASES OF UNKNOWN ORIGIN

*Etiology of Köhler's Disease.*—Valtancoli [17] discusses various theories as to the cause of Köhler's disease of the second metatarsal bone, reporting four cases. He considers that either violent trauma or the trauma of strain plays an important part, adding that Köhler gives the following as being characteristic of the disease of the second metatarsal bone that bears his name: the distal half of the second metatarsal bone

17. Valtancoli, G.: Chir. d. org. di movimento **9**:447 (July) 1925.

is increased in volume from the center toward the extremity so that the metatarsal neck is completely absent. The metatarsal head is shortened, as if it were depressed. The articular surface of the head, having lost its convexity, is flattened in recent cases and in older cases presents irregular tubercles and depressions. The articular surface of the corresponding proximal phalanx becomes irregular.

*Vertebral Epiphysitis: A Case.*—Lewin [18] reports a case of vertebral epiphysitis. There are two epiphyses for each vertebral body, one at the upper pole and one at the lower. These unite with the body at about the eighteenth year; therefore, epiphysitis cannot occur after this period. However, before this period of fusion occurs the vertebral epiphyses are subject to the same conditions as are epiphyses in general, first, infection, local and remote; second, circulatory disturbance in the nature of embolism, thrombosis, etc.; third, traumatism both internal and external, and, fourth, glandular disturbance affecting bone growth and development. The signs of an epiphysitis are similar to those of an early tuberculosis: limitation of motion, muscle spasm, military attitude, pain, night cries, tenderness and sensitiveness to jarring. Anteroposterior and lateral roentgen-ray views should be made in order to reveal characteristic involvement in the region of the epiphysis. Lewin thinks that vertebral epiphysitis is a definite pathologic entity, and is analogous to Legg-Calvé-Perthes' disease of the hip; to Osgood-Schlatter's condition in the upper tibial epiphysis; to Köhler's tarsal scaphoiditis; to Freiberg's infraction of the metatarsal head, and to apophysitis of the os calcis.

*Vertebral Epiphysitis: A Cause of Spinal Deformity.*—Buchman [19] reports five typical cases of this condition. In an examination of the roentgenograms of 120 unselected cases of various spine infections, he has found that fifty-three out of sixty-five idiopathic curvatures show positive evidence of this type of epiphyseal disease. The remaining fifty-two cases, which were negative, included poliomyelitis, ostearthritis, rickets, and tuberculosis. Normally, the superior and inferior vertebral epiphyses appear at about 11½ years of age. The pathologic changes seen in the roentgenogram are as follows: The epiphyses become enlarged, moth-eaten, frayed and indistinct; the intervertebral spaces become cloudy, mottled and irregular; the vertebral outlines become indistinct. All of these parts then tend to fuse into one mass. This is followed by a stage in which the process is reversed. Between the ages of 10 and 21 symptoms of fatigue, backache, tenderness of the epiphyses and characteristic roentgenographic changes then make their appearance. Many of these patients develop deformities of the spine. The etiology

18. Lewin, P.: Ann. Surg. **82**:286 (Aug.) 1925.
19. Buchman, Joseph: J. Bone and Joint Surg. **7**:814 (Oct.) 1925.

is unknown. There is evidence leading to the belief that it is a low grade infection in the spine. Stress and strain constitute additional factors which aid in the production of deformity.

*Juvenile Osteochondritis of the Spine.*—Valtancoli [20] reports three cases of what he calls juvenile osteochondritis of the spine. In the roentgenograms he followed the development of the disease from its beginning to its seemingly finished state. In the first case reported the disease was in its initial state and the lesions were exclusively in the epiphyseal disks, which showed thickening and alteration in their form, with an irregularity of the line of demarcation between the disks and the body of the vertebra. The kyphotic curve was not pronounced, and as a consequence there was little deformity of the vertebral bodies. In the second case the lesions were more advanced. The deformity had been noticed clinically for more than a year, and the alterations seen in the roentgenogram involved not only the epiphyseal disks but also the vertebral bodies, which had assumed a more or less marked wedge shape. Nevertheless, the intervertebral spaces were well preserved. In the third case the form of the bodies was more regular, but they appeared to be crushed in their ventral half and had assumed an aspect decidedly cuneiform, although the intervertebral spaces were not lessened. The author states that the roentgenologic picture is too characteristic to allow of an error in diagnosis in the case of the trained observer, but it is necessary for recognition that the roentgenogram be taken laterally. At the beginning, differential diagnosis from tuberculous spondylitis might present difficulty. Clinically, the diagnosis is based on almost complete absence of pain, although there is marked rigidity in the kyphotic area. It is based also on the negative biologic reactions, and above all on the roentgenogram in which the epiphyseal lesions are shown. The benign character of the disease and its roentgenologic appearances suggest that this condition be included in the chapter of juvenile osteochondritis described by Scheuermann as deforming juvenile osteochondritis of the back. The treatment consists of early correction; later it is exceedingly difficult to change the deformity.

[ED. NOTE.—These articles on wedge shaped vertebrae suggest that observations made on the lateral roentgenograms of spines will bring to light the probable frequency of this condition, unwarrantable as it is at present to attempt to establish a clinical entity before further study of this condition.]

*Observations on Osgood-Schlatter's Disease*—Ollerenshaw [21] says that as a result of personal investigation and treatment of thirty-two cases, Osgood-Schlatter's disease is, he believes, a condition of purely

20. Valtancoli, G.: Riforma med. **41**:823 (Aug. 31) 1925.
21. Ollerenshaw, Robert: Brit. M. J. **2**:944 (Nov. 21) 1925.

traumatic origin, of definite pathology, demanding a certain line of treatment, and that it shows some clinical variations. He believes that the condition should be treated as a fracture, by complete immobilization for six weeks in a well fitting plaster cast from the groin to the ankle. Walking is permitted, and, afterward, a knee cage limiting motion to 45 degrees for an additional six weeks. In his series, more than half (nineteen) were in girls, while Osgood's original ten cases were all in boys.

[ED. NOTE.—Repeated plaster strapping is sufficient treatment in most cases in our opinion.]

*Coxa Plana.*—Wideroe [22] reports a case of coxa plana in which the patient was studied by roentgen ray every month for three and one-half years. On the basis of roentgen-ray appearances he divides the condition into two stages: a stage of destruction and a stage of reconstruction. In the case he reports, the first stage lasted for a year and a half; the second, at the time of the report, had lasted two years.

*Roentgen-Ray Study of Nine Hundred and Twenty-Six Cases of Rickets.*[23]—Nine hundred and twenty-six children were studied roentgenographically by Grover, Christie and Merritt and 636 (66.5 per cent) were found to have rickets in some form. Of the white children studied, 59.2 per cent. were rachitic; of the black, 79.7 per cent were rachitic. Florid rickets in the white were 9.2 per cent, and in the black, 21.5 per cent. By clinical methods of examination used in 1,439 children, 68.7 per cent had rickets. The writers feel that these figures are practically identical, and it is of great interest that they were secured from two such widely separated angles. Therefore, they believe that for the purpose of establishing a diagnosis of rickets, the roentgen ray offers more dependable data than can be secured by a clinical study. This is especially true of the florid type.

*The Control of Rickets: Preliminary Discussion of the Demonstration in New Haven.*—In carrying out an interesting experiment in New Haven, Eliot [24] has come to feel that a slight degree of early rickets is well nigh universal in our climate and state of society. The intimate association of rickets with growth, its early appearance regardless of season, and its universality raise the question whether this slight degree of rickets must not be considered as normal. The author is not prepared to answer this question. It was found that the diagnosis of rickets could be made earlier by roentgen ray than clinically. Clinically, the diagnosis was made in 80 per cent, the earliest being four months, the

22. Wideroe, S.: Norsk. Mag. f. Lægevidensk., August, 1925, p. 811.

23. Groover, T. A.; Christie, A. C., and Merritt, E. A.: Radiology **5**:189 (Sept.) 1925.

24. Eliot, Martha M.: J. A. M. A. **85**:656 (Aug. 29) 1925.

average being eight months. Roentgenologically, 53 per cent of the diagnoses were made in the third and fourth months. The importance of this can be appreciated from a preventative point of view.

*Osteitis Fibrosa Cystica.*—According to Sisk,[25] this condition is a distinct, benign, central lesion of the bone. The uniformly good results obtained by conservative surgery in cases of osteitis fibrosa cystica make this an important disease to be considered in cases of central bone lesion. The etiology is not definitely known, but trauma appears to be a common factor. The pathogenesis appears to be that of metaplasia and early malacia. Diagnosis is made by the roentgen-ray examination, supported by a long history with onset in early life, and, if there is no fracture, little or no loss of function. Differential diagnosis is to be made between osteitis fibrosa cystica and other common central lesions. Microscopic examination of tissue removed is essentially important in making a prognosis. If the patient is confined to bed many months, some of these lesions heal spontaneously, as was suggested by Bloodgood recently. The uniform success obtained by surgery, with a comparatively short convalescent period, however, argues against this. If the cystic area is small, it is sufficient to expose the tumor, break through the thin canopy, and thoroughly clean out all soft tissue with a curet. Following this procedure, the tumor stops its growth, as do benign cysts of other tissues when their contents are evacuated and the cystic capsule is removed. The cortical canopy should be crushed in, and if bone chips are introduced into the cavity, healing is hastened. Except in cases of extensive involvement with deformity, efforts at plastic surgery are not indicated.

*Osteomalacia in China.*—Maxwell and Miles[26] report a complete study of osteomalacia in China (Pingtingchow, Province of Shansi). The subject is discussed under the following headings: history and distribution; general picture of the disease; the fetus in cases of maternal osteomalacia; etiology; the sequelae of osteomalacia; treatment, and osteomalacia in animals. The author's general conclusions are: China is at present one of the world centers for the study of osteomalacia. In the special area involved, the incidence is from 1 to 3 per cent of child-bearing women. While its effects are more often shown in pregnancy and the puerperium, it frequently begins about the time of puberty. The disease is one due to a deficiency diet, the principal lack being in a shortage of mineral content and the activator for calcium metabolism. Lack of sunlight and movement are contributing factors in its production. The bones of full term children from mothers with osteomalacia do not show the signs of rickets, but of osteoporosis. There is, however,

25. Sisk, J. N.: Surg. Gynec. Obst. **41**:481 (Oct.) 1925.

26. Maxwell, J. P., and Miles, L. M.: J. Obst. & Gynec. Brit. Emp. **32**:433, 1925.

evidence to suggest that these infants develop rickets more easily than the normal child. The sequelae of the disease are twofold: one, deformity of the pelvis, the results of which on labor may be counteracted by a planned cesarean section; two, interference with marital relations, which may be treated by a new operation in which portions of the pubes and ischium are resected. The ovaries have nothing to do with the disease, and it is not necessary either to remove them or to tie the fallopian tubes, provided that the woman is treated with cod liver oil and calcium, and this treatment is kept up during any future pregnancy. By improving the diet and supplying cod liver oil, it should be possible in due time to stamp out the disease. Prevention is the principal thing, and to this end a settled government and improved means of communication are essential. Animals also suffer from this disease, and can be cured by the administration of cod liver oil.

### NEOPLASMS

*Bone Tumors: Analysis of One Thousand Cases.*—Christensen [27] in this analysis of 1,000 tumors follows the classification of the committee of the American Pathological Society and the College of Surgeons. It includes benign osteogenic tumors, thirty-five, and malignant, 441; periosteal fibrosarcoma, six; giant cell tumors, 362; myelomas, twenty-eight; endothelial myelomas, eighty-seven, and metastatic tumors, thirty-three. The sites of predilection for osteogenic and giant cell tumors are at the ends of long bones where there is a disk of "maximal growth"; this is the point at which the growth period is longest and the natural growth momentum greatest. Then, too, it is to be observed that the localities in which most malignant tumors occur are also the seat of the benign tumors. From this analysis the following summary may be made: Of the osteogenic sarcomas, 58.7 per cent occur in the male, and 41.3 per cent occur in the female. The highest age incidence for osteogenic sarcoma, for both sexes, is in the second decade of life, although more than 33 per cent occur after the thirtieth year. The epiphyses are not involved in at least 33 per cent of the cases with involvement of the diaphyseal ends of long bones. The epiphyses are invaded in 90 per cent of the giant cell tumors involving the ends of long bones. Both sexes are about equally affected with giant cell tumors; namely, female, 73 per cent and male, 47 per cent. Giant cell tumors are most common in the third decade of life, but 32 per cent occur after the thirtieth year. Multiple myeloma is not a disease limited to adult life; 39 per cent occur before the twenty-first year. Benign angiomas must be considered in every diagnosis of a vascular tumor of the bone. Solitary diffuse endothelioma is not an uncommon tumor, especially in younger people.

27. Christensen, F. C.: Ann. Surg. **81**:1074 (June) 1925.

Predisposing sites for benign and malignant osteogenic tumors and giant cell tumors are at the ends of long bones, where there is an epiphyseal disk of maximal growth, where the growth period is longest, and where the natural growth momentum is greatest.

*Multiple Myeloma.*—Meyerding [28] reports thirteen cases of multiple myelomas from the Mayo Clinic, and says that he believes it is important to bring out the vague symptoms presented, which usually suggest rheumatism or neuritis. It is obvious that the roentgen ray is the greatest aid in diagnosis. However, the presence of large amounts of albumin and Bence-Jones protein in the urine, together with vague symptoms of multiple myeloma, are suggestive.

*Metastasis to the Bones from Carcinoma of the Breast: A Roentgenologic Study.*—Meyerding [29] has reviewed 1,985 cases of carcinoma of the breast. In sixty-seven (3.48 per cent) metastasis to the bones had occurred. Necropsy records have shown percentages as high as 26.5 per cent of bone metastasis. This is doubtless too low, as all bones are not examined at necropsy. It is fairly certain that with carcinoma of the breast there is a higher incidence of metastasis to the bones than with carcinoma in any other organ. In Meyerding's series, metastasis was found in the pelvis of 38 per cent; in the femur in 34 per cent; in the lumbar spine in 31 per cent; in the ribs in 26 per cent; in the dorsal spine in 16 per cent, and in other bones much less often. These statistics were made from roentgen-ray findings in living patients, and they differ, therefore, from necropsy findings. When the pelvic ring was involved the lesions were about equally divided between the ilium, the ischium and the pubis, the sacrum being implicated alone in only four. The femur involvement was usually in its upper third. In the spine it was confined to a single vertebra in only four cases, and the lumbar region was involved in 58 per cent. He believes that efficient surgery has a decided effect in postponing metastasis to the bone. From his findings, which he feels are insufficient to make a final statement, radiotherapy was of no effect in slowing metastasis—indeed, it had the opposite effect. The metastatic lesions are commonly of the osteoplastic type, and are characterized roentgenologically by a honeycombed appearance, with a marked decrease of density and mottling in the affected areas.

*The Treatment of Bone Sarcoma by Toxins, Radiation, Amputation or Resection.*—Bloodgood [30] answers the question, "Has the toxin treatment of Coley alone accomplished a cure without amputation or resection?" by saying that it seems to be the opinion of the Committee on

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28. Meyerding, H. W.: Radiology **5**:132 (Aug.) 1925.

29. Meyerding, H. W.; Carman, R. D., and Garvin, J. D.: Radiology **5**:486 (Dec.) 1925.

30. Bloodgood, J. C.: Am. J. Roentgenol., September, 1925, p. 253.

Registration of Bone Sarcoma of the American College of Surgeons that there is only one case in which the cure may possibly be credited to the toxins alone. It is more difficult to answer the question, Is there a larger percentage of five year cures in the cases of sarcoma of bone in which in addition to resection or amputation the toxins have been used? Bloodgood himself has given up the use of toxin, but he hopes that Coley and others will continue it, as it is worth while finding out whether it really does increase the probability of a cure. Because of the difficulty of diagnosis at exploratory operation, Bloodgood follows the rule not to amputate if there is any doubt as to malignancy. Resection, with bone transplantation, may be done. This rule is based on the fact that in early cases the chances are that the lesion is benign, and at the present time the probability of a cure by amputation is too small to justify this procedure, unless the diagnosis is certain.

*Fibroma of the Tendon Sheath of the Deep Flexor of the Right Ring Finger.*—Avoni [31] reports a case of true fibroma of the tendon sheath of the flexor profundus tendon of the right ring finger of a woman aged 64. It had been present fifteen years and had caused no inconvenience, except that its presence interfered with the use of farm implements. Microscopically it was completely encapsulated and was made up of mature connective tissue cells. He says it is a rare tumor developing from a tendon sheath.

### OSTEOMYELITIS

*A New Therapeutic Sign in Osteomyelitis.*—Hedri [32] calls attention to a sign which he believes to be of considerable importance in determining the extent to which surgical intervention should be carried in cases of acute osteomyelitis. This is lipuria or the presence of fat droplets in the urine. Apparently, it occurs when pus has collected in the medullary space under a certain pressure. It is therefore to be interpreted as a sign of extensive infection and an indication for thorough opening up of the medullary canal in contrast to the method of draining only the subperiosteal abscess or of making a small opening into the metaphyseal portion of the bone.

*"Silent Foci" of Osteomyelitis.*—Brickner,[33] discussing what Phemister has termed "silent foci" of localized osteomyelitis, favors the view that these represent dormant localized infections which occurred at the time of the acute hematogenous invasion rather than late metastases from an active focus not yet extinct. He believes that they represent attenuations of the original infection and that conservative operative procedures

31. Avoni, Aldo: Chir. d. org. di movimento **9**:629 (Oct.) 1925.
32. Hedri, A.: Zentralbl. f. Chir. **52**:1412 (June 27) 1925.
33. Brickner, W. M.: J. A. M. A. **85**:1782 (Dec. 5) 1925.

should be employed in their treatment, such as drill drainage of chronic bone abscesses, simple sequestrectomy, simple drainage of subperiosteal abscesses, etc. Clinical data are presented in support of these views.

*A New Biologic (Cultural) Method of Treatment.*—Thorek,[34] at the American Hospital, Chicago, for the last three years has been making an experimental and clinical investigation of the action of aluminum potassium nitrate in the treatment of chronic infections. The results have proved so satisfactory that he feels that the method is now worthy of more extended use. He was led to the present chemical agent through earlier trials of potassium nitrate, which from observation in the meat preserving industry and from some scattered reports in the literature was known to be an effective oxidizing agent. Potassium nitrate proved to be irritating, and after extended search the nontoxic, nonirritating aluminum potassium nitrate was selected as best meeting the requirements. Animal experimentation shows that the compound is entirely nontoxic when given orally, intravenously and subcutaneously in even much higher dosage than when employed clinically. It is prepared by adding aluminum nitrate and potassium nitrate in the proportion of the molecular weight of one molecule of the former to three molecules of the latter, in sufficient hot concentrated nitric acid to dissolve the substances. On cooling, the double salt crystallizes out. The product is further diluted with nine parts of potassium nitrate and the resulting mixture is incorporated in the dressing as described below. The action of the aluminum potassium nitrate compound was found, both experimentally and clinically, to be quite different from that of antiseptics and germicides. It assists and intensifies the growth of bacteria when added to culture mediums and it tends by rapid propagation to lower the virulence and viability of the infecting organisms. It is used in the form of a plastic dressing applied directly to the skin. Rolled oats, sterilized for two hours to destroy the proteolytic enzymes, is employed as the vehicle. This is prepared by adding 50 cc. of boiling water to the ounce of dry rolled oats and stirring until a uniform mass is produced. The average dose is approximately 13 Gm. of the compound to the ounce of dry rolled oats. The dressing is applied about one-eighth inch (0.3 cm.) thick, directly to the skin, well beyond the limits of the affected area, and is covered over with waterproof material such as oiled silk. It should be changed at least once in thirty-six hours. The first effect and typical reaction is the appearance of an erythema within forty-eight hours. This is followed by the appearance of pustules and vesicles containing purulent fluid confined to the area affected. If applied to an area in which there is no disease, no reaction appears. There is a great

34. Thorek, M.: Illinois M. J. **48**:112 (Aug.) 1925; M. J. & Rec. **122**:132 (Aug. 5) 1925.

increase in the amount of discharge from the sinus and this contains many leukocytes and organisms. If the dressing is discontinued the discharge diminishes and again reappears with its application. Pain is markedly relieved. Although effective in all suppurative processes, the most striking results have been obtained in chronic osteomyelitis. Of 116 patients so treated from 60 to 70 per cent have been discharged cured; 10 per cent have improved, and from 14 to 17 per cent have been unchanged. It is not claimed that the method will do away with the necessity of operation in most cases, but that it will render such procedures more effective and that it represents a powerful therapeutic adjuvant.

[ED. NOTE.—We have seen an article as yet unpublished by Strauss, Kreuscher, Greunerud and La Porte in which the results of other experimental and clinical work with this same substance are reported. Their experiences provide striking confirmation of Thorek's claims. We believe that the method should be tried and the results reported.]

*(To be continued)*

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